THE PNEUMONIA COMPLEX IN BIGHORN SHEEP

by

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The declining bighorn sheep population in the United States and Canada is the result of many things. One major decimating factor has been encroachment of historic ranges by man and domestic animals. Another major catastrophic factor has been lung diseases. Both are well documented. The latter may be assumed to be one of the results of the former.

Major catastrophic die-offs from disease have been said by many researchers to have been caused by pneumonias of one form or another and, in the case of the early part of this century, major losses in the western United States were attributed to scab (Ward 1915). The scab was presumably *Sarcoptic* mange of domestic sheep. There is some doubt in my mind about the validity of scab as being the true cause of these losses. Reports of the incident stated "sheep died in such numbers that their bodies clogged small streams." A man who was in the back country of Idaho at this time once heard my description of pasteurellosis in these animals and saw color photographs of the gross symptoms. He then told me that "this was what the insides of sheep thought to be dying of scab looked like -- they seemed to be rotten inside very shortly after death."

The cause of many of these decimating losses among bighorn sheep is lost to us forever because complete bacteriological, virological and parasitological studies were not made. The written accounts are all that is available to us today. We must take these written words at face value and attempt to use them for finding answers to present day problems.

We do know there is much documented evidence that pneumonias are very prevalent in bighorn sheep. These animals appear to be more prone to lung ailments than to any other single disease factor. We know that most of our bighorn sheep populations are not plagued to any great degree by predation, accidental deaths attributed to living in precipitous habitats, etc. (Honess and Frost 1942, Moser 1962, Smith 1954). These factors do take their toll, but the pneumonia complex appears to be of prime importance.

Nearly each of the major writings on bighorn sheep in the last 50-75 years note pneumonia problems as a mortality factor. Some of these make a summation of symptoms and give thoughtful diagnosis of the cause of mortalities. Many of these give inconclusive evidence of the correctness of the diagnoses.

The pneumonia complex we speak of in bighorn sheep may be defined as the relationship of the various pathogenic organisms in the lungs and respiratory tract, to the pathology in the specific organ in which they

are found. We know of two major entities being present in bighorn sheep respiratory tracts: the various bacterial species and the various nematode species. The *Mycoplasma* have been isolated from captive bighorn sheep - not from wild ranging bighorn sheep (Woolf et al. 1970). Attempts have been made to isolate viral organisms from bighorn sheep mortalities. Results have always been negative. We can only postulate the presence of viral pathogens and *Mycoplasma* as being responsible for some pneumonic complications in these animals in the wild state.

We do know much about the various bacteria isolated from organs of the respiratory tract of these animals. We also know much about the lung nematodes specific to these animals.

The bacterial and the nematode entities have probably been associated with bighorn sheep for eons. No doubt the nematodes came onto this continent with the ancestors of present day bighorn sheep. We can trace this back to similar species of lung nematodes found today in wild sheep of Asia (Boev 1950). The original source of the bacterial entities is more difficult to trace with certainty. Some of these bacteria are similar or identical to bacteria found in domesticated sheep or other domesticated animals throughout the world. Similar or identical *Pasteurella* and *Corynebacterium* species can be found in domestic sheep or cattle. These organisms can also be bound in elk or pronghorn antelope.

Species of bacteria other than the genus *Pasteurella* or *Corynebacterium* have been isolated from normal and pneumonic bighorn sheep. Some of these belong to the genus *Staphylococcus*, *Streptococcus*, *Nisseria* and *Diplococcus*. The relationship of these to the pneumonia complex in bighorn sheep is largely unknown.

Some authors have referred to the pneumonia complex in bighorn sheep and leave the reader to believe the major cause is lung nematodes of the genus *Protostrongylus* (Forrester and Senger 1964, Beuchner 1960). These parasites must be taken into consideration in studying the pneumonias of bighorn sheep. They probably are not an all-encompassing cause of the problem though.

There are two recognized species and one species of questionable recognition of the genus *Protostrongylus* in bighorn sheep on our continent. These nematodes appear to be specific to the bighorn sheep species of North America. One or the other or both species are found in most wild ranging bighorn sheep. All animals in some flocks harbor these lung nematodes. I have never found a bighorn sheep that did not shed *Protostrongylus* first stage larvae in their feces. Sometimes collection of feces for several consecutive days was necessary to prove that the animal was infected.

There appear to be at least three pneumonia types in the pneumonia complex of bighorn sheep. The first type I will discuss is the very acute, very devastating pneumonia associated with *Pasteurella* infections. This pneumonic syndrome very often terminates rapidly into septicemia. Once septicemia develops, the animal's death is usually inevitable. This type of pneumonia may be brought on by the normal presence of *Pasteurella*

bacteria in the respiratory tract and some type of stress; physical, physiological, psychological. The presence of animals which have been so stressed and are now shedding virulent (possibly capsulated) *Pasteurella* in nasal and throat discharges may also be a source of infection. The infection develops rapidly, causing acute consolidation and hepaticization of lung tissue, highly hemorrhagic nasal turbinates and trachea, and invasion of the blood stream from these areas. The organisms spread rapidly throughout the body causing extreme cyanosis of all organs and tissues. *Pasteurella* (usually either *P. multocida*-like or *P. hemolytica*-like) organisms can be isolated from almost all body tissues at this time. Death usually occurs very quickly after septicemia develops (Post 1962).

A second type of pneumonia is associated with presence of lung nematodes. This usually develops as a chronic condition. The lung nematodes cause tissue reaction around the site where the worm occurs (either in parenchymal tissue or attached to the lining of the bronchioles). Tissue reactions involving *P. stilesi* in the parenchymal tissue include alveolar collapse, loss of alveolar structure and scarring. Female *P. stilesi* release eggs which soon hatch into first stage larvae. Eggs that die before hatching, for one reason or another, and first stage larvae which die, for one reason or another, cause much tissue reaction. Some cases show increases in phagocytic leucocytes, formation of giant cells (sometimes referred to as foreign body cells) and fibrocytic invasion. Living larvae do not appear to cause these tissue reactions to any great degree. Living larvae wander through the lung parenchyma until they reach open alveoli or very small bronchioles. They may irritate bronchiolar linings to some extent, especially so if they die and disintegrate at this time. Affected bronchioles may show thickening of mucosal linings and increased mucous exudate. The exudate and larvae move out into larger bronchioles and to bronchi where they are swept from the respiratory tract.

Presence of *P. rushi* in the bronchioles or bronchi do cause some irritations to mucosa which in turn causes increased mucous exudate. First stage larvae of *P. rushi* are picked up in mucous and swept from the respiratory tract. The productive cough of verminous pneumonia may be associated with presence of excessive amounts of mucous and first stage larvae being removed in this way.

Terminal verminous pneumonia could be associated with large numbers of lung nematodes which cause parenchymal tissue destruction and scarring, inability of the animal to cope efficiently with its environment, loss of body weight to emaciation in some cases and a racking productive cough. Death usually occurs as a slow debilitating process.

A third type of pneumonia may occur from other bacteria (*Corynebacterium* sp., *Staphylococcus* sp., *Streptococcus* sp., *Diplococcus* sp., *Nisseria* sp. or others). Certain of these may cause purulent abscesses in lung tissue. This type of pneumonia is usually chronic to sub-acute in nature. The disease usually develops slowly by destruction of alveolar and broncheolar structures. These structures are replaced by masses of polymorphic and lymphocytic leucocytes, sometimes plasma

cells and giant cells. This is a typical inflammatory response. Progression of this type of pneumonia, sometimes referred to as bronchial pneumonia, causes loss of normal lung function as well as symptoms of presence of bacteria and their toxins. If death occurs, terminal symptoms are labored and shallow breathing, a productive cough and general appearance of illness.

Understanding the entire pneumonia complex in bighorn sheep requires a knowledge of these three types of pneumonia because presence of one type may not be there without evidences of the other. Pasteurellosis symptoms in the lungs of these animals is usually accompanied by signs of verminous pneumonia. Bronchial pneumonia is also usually accompanied by signs of verminous pneumonia. Certain literature references to pneumonia symptoms in bighorn sheep mortalities indicate that usually two and sometimes all three types of pneumonia were present in the animals. This is much of the reason why recorded diagnostic information on pneumonia in bighorn sheep is so confusing. Add to this the possibility that *Mycoplasma* and viral organisms may also be complicating factors and the observer or diagnostician may be doubtful of the major cause of mortality. I have seen very few cases of pneumonia in bighorn sheep which I felt were a disease syndrome with uncomplicating factors.

Examination of the pneumonia complex in bighorn sheep with the above in mind will show the complicated nature of the disease. Holding of bighorn sheep in captivity so that this complicated disease can be studied experimentally is not possible at the present time. Usually spontaneous pneumonia or pneumonia-septicemia interferes with the study. Antibiotics used to control the syndrome alter the animal so that it loses its value as an experimental animal. We need a laboratory animal with a physiological similarity to bighorn sheep in order to solve problems. Attempts to infect domestic sheep with the *Protostrongylus* species have failed (Post 1958, Post and Winter 1957). Therefore, this animal cannot be used as a direct corollary to the bighorn sheep to measure the contribution of lung nematodes to the pneumonia complex. Development of the unique syndrome of pasteurellosis in domestic sheep is difficult because the same behavioral and psychological stresses cannot be placed on a domesticated sheep as would occur to a wild or semi-wild bighorn sheep. Our greatest need, then, is to find a method of holding relatively normal bighorn sheep in captivity. Only then will we be able to give careful and controlled attention to the diseases that plague these animals, to normal biochemistry and to all the biological, physical and behavioral factors which make this animal unique to its environmental niche on our planet.

LITERATURE CITED

Beuchner, H. K. 1960. The bighorn sheep in the United States, its past, present and future. Wildl. Monographs. 4:174 pp.

Boev, S. N. 1950. Systematics of lung nematodes of the genus *Protostrongylus* Kamensky, 1905. Akad. Nauk. S.S.S.R. 4:64-67. In: Helm. Abstr. 19(5):29.

Forrester, D. J. and C. M. Senger 1964. A survey of lungworm infection in bighorn sheep in Montana. J. Wildl. Manage. 28(3):481-491.

Honess, R. F. and N. M. Frost 1942. A Wyoming bighorn sheep study Bull. No. 1. Wyo. Game and Fish Dept. Cheyenne. 127 pp.

Moser, C. A. 1962. The bighorn sheep of Colorado. Colo. Game and Fish Department, Denver. 49 pp.

Post, G. 1962. Pasteurellosis of Rocky Mountain bighorn sheep. (*Ovis canadensis canadensis*) Wildl. Dis: WD-62. No. 23.

Post, G. 1958. Life Cycle of lungworms. F. A. Wyoming. Project No. FW 3-R:58-61.

Post, G. and K. B. Winter 1957. Life Cycle of lungworms. F. A. Wyoming Project No. FW 3-R:48-52.

Smith, D. R. 1954. The bighorn sheep in Idaho. Wildl. Bull. No. 1 Idaho Dept. of Fish and Game, Boise. 154 pp.

Ward, H. 1915. Octariosis in the bighorn. J. Parasitol. 1:121-127.

Woolf, A., D. C. Kradel and G. R. Babash. 1970. *Mycoplasma* isolates from pneumonia in captive Rocky Mountain bighorn sheep. J. Wildl. Dis. 6(3):169-170.

DISCUSSION

QUESTION BY C. E. WILLIAMSON, USFS, COLORADO: With herd removal of bighorn can the lungworm life cycle be interrupted successfully so that a clean herd can be restored to that area?

REPLY BY POST: There have been several tests to try to remove the lungworm from bighorn sheep. One of our speakers this afternoon will mention Tramisol but we still haven't found a good drug to completely remove the lungworm from bighorn sheep. This is a good objective. When we do transplanting operations we must have something so we can put a clean herd back into a historic range.

QUESTION BY DICK PILLMORE, BSF & W, COLORADO: I agree with most of what you said, that it is a pneumonia complex, but in the same herd you can have a whole spectrum from the acute to the chronic situations showing up. I think George can remember the animals that Glen Eyrie had in Colorado Springs that had acute infections where the animals were still fat with a lot of edema in the lungs. Others had great abcesses and some, particularly lambs, had very heavy lungworm infections. We really had the whole spectrum.

REPLY BY POST: That means it is a pneumonia complex. When you get all three of them you should not call it just a lungworm-pneumonia complex, should you? You should call it a pneumonia complex.

DISCUSSION FOLLOWING PANEL

COMMENT BY PARRY LARSON, G & F, NEW MEXICO: In regard to base levels in phosphorous, a good many western states are, from a livestock standpoint, in acknowledged phosphorous-deficient zones. Did any of you who are doing work on this get base levels for phosphorous? Do we know what the normal status is in bighorn? Is it related to phosphorous-calcium ratios as they normally establish in domestic livestock? Is this kind of work being done or can it be done?

REPLY BY GEORGE POST, CSU: There has been a little done in the past here in Colorado. Robert Streeter, a Ph.D. candidate about two years ago, worked on the Buffalo Peaks herd. He did calcium-phosphorous ratios on plants that he collected in bighorn sheep ranges and he tried to correlate these with demography to some extent. But again, he didn't have anything on requirements of the sheep itself.

REPLY BY LARSON: I was interested because we were working three or four years ago on pronghorn and were taking some spring blood samples. We were trying to get vitamin A, carotin, and calcium:phosphorous ratios. We sent them to CSU and we got levels which were extremely high compared with normal levels of sheep or livestock. This seems like a terrific opportunity for the talents of the people we have here. It is something that can probably be evaluated from field samples and we could know something about seasons or, any time we wanted to get samples, to see if things like vitamin A levels or calcium-phosphorous levels are probable deficiencies.

REPLY BY POST: Of course vitamin A is very fragile. It would be very difficult for you in New Mexico to take samples and send them here for analysis. Calcium and phosphorus, yes, field samples can be sent in for data.

We especially need this on normal animals. Our big problem is that the only time we get our hands on an animal is when it is sick. Also, if we put a fairly normal animal under confinement he also becomes sick. We use antibiotics on him and then he is no longer a normal animal. If we could get normal, wild sheep samples, this would be one way of building up knowledge. If we use a Cap-chur gun on an animal it takes him five or six minutes to go down. Is he normal? Consider some of the things that happen to an animal when he is frightened. The spleen squeezes up, putting new blood into circulation - blood that has been stagnating in the spleen. A new shot of adrenelin comes in.

Consider a trapped animal. You trap him at 4:00 in the morning or just at daylight and you don't get your hands on him for half an hour. He is not 100% normal either. This is our real problem. We don't have normal animals to work with and we don't have access to normal animals.

COMMENT BY AL FRANZMANN, UNIVERSITY OF IDAHO: I want to follow up on that. This is the whole object of the study I have been doing - to try to get some baseline values of phosphorous. In all the wild sheep,

almost 100, the phosphorous level very seldom went above 4 milligrams percent which is about half of what domestic sheep would hope to have. Not only that, our calcium:phosphorous ratios were 3:1. We studied a group of 80 captive sheep which were on good nutrition. The diet was supplemented with protein and free choice minerals, not only calcium-phosphorous, but also trace minerals. We got values that we expect in domestic sheep. So we can relate to our domestic species in this case and get a pretty good idea of what we should expect on bighorn.

REPLY BY POST: These wild animals that you were using, were they exercised before you got your blood samples?

REPLY BY FRANZMANN: Yes. Most of them were in some of the higher excitability classes. It so happened that there are mineral excitability classes as well as blood urea nitrogen excitability classes. But this was not statistically significant in affecting calcium-phosphorous levels.

REPLY BY POST: This was kind of strange because when you exercise an elk, its serum phosphorous just goes down. A group of elk were being worked in a trap. I was taking blood samples at intervals so I could get an age classification and a sex classification. I took my first sample when the group was very fresh in the trap and my last sample when they had been in for an hour or so. We could just see this nose-dive in phosphorous with exercise. By the time the last one came through, he didn't have enough phosphorous to hold him on his feet. This would be a state of shock and in a state of shock like this, the phosphorous was the only thing that really showed up significantly. In the early part of the day the animals had 11 to 12 milligrams percent, but this dropped to 2 milligrams percent in the one that collapsed under this exercise stress.

REPLY BY FRANZMANN: How many individuals?

REPLY BY POST: There was a series of 12; one taken over a period of about an hour or hour and a half.

REPLY BY FRANZMANN: We did the same thing with sheep using M-99 which produces no excitement phase. Later samples were taken after handling, after being held overnight and after two weeks. We found the same levels throughout. You can only report what you find. I am sure when you reach a certain level you get this change that is effected because you are in a condition of extreme stress. These are things that we have to measure, though.

COMMENT BY DARYLL HERBERT, OF BRITISH COLUMBIA: I have been monitoring phosphorous in plants and animals - in bighorn sheep - for a year now. I monitored the cycle in the winter range plants every month from the growing season through the following growing season. I also did this in alpine range plants. I found adequate phosphorous levels according to NRC requirements for domestic cattle and sheep which I feel are probably higher than we can use for bighorn sheep. I found levels in winter range plants of 2600 to 2800 ppm. This declined throughout the year to about 500 ppm the following spring prior to growth. The subalpine and alpine

plants range from about 2000 ppm in medium phenological stages of subalpine growth to over 3000 ppm until September. This appeared adequate for lactation. It was above the lactation levels for domestic sheep so I am sure it was above the lactation levels for bighorn sheep.

We monitored blood phosphorous levels throughout the winter from September through the following spring. The phosphorous at that time ranged from about 800 ppm in September down to about 500 ppm throughout the winter. This is below any winter requirements that anybody else has found, notably Watkins and Hoax (?) in New Mexico in the 1930's and 40's. We didn't find very drastic changes in the blood phosphorous. They ranged from between 4 and 7 milligrams percent which, again, is supposedly normal for domestic sheep. After the one-year period we also fed the high quality, pelleted ration which contained about 6600 ppm phosphorous. This brought the blood phosphorous level up to almost 10 milligrams percent. So this correlates well from the high level to the low level. I think that eventually I missed out on part of the year's cycle with the blood phosphorous, but I think that we can get a good correlation between the blood phosphorous and the forage phosphorous so that we can use it as a field method. I haven't found that the bighorn sheep is deficient or unable to get sufficient phosphorous from the range, at least in British Columbia.

COMMENT BY STEVE HAWKS, BLM, IDAHO: Has anything been done as far as investigating the bighorn sheep and parasite problems, especially lungworm, from an evolutionary point of view? Have they evolved in such a way as to prevent elimination of the host?

REPLY BY GEORGE POST, CSU: If you are going to have the worm, you must have the host. If you don't have an adequate host, the worm isn't going to be here either. You can search the literature on where the parasite came from. It probably came over with the early sheep that came over the Bering Strait to Alaska. Somehow it has been able to survive in the bighorn sheep during all these eons of time. It's been able to adapt and this, of course, is the indication of a successful parasite.

REPLY BY HAWKS: How about this excitement factor? It seems that this pneumonia complex problem really crops up during a stress. Maybe the parasite has developed itself not to become virulent until the stress factor reaches a certain point.

REPLY BY POST: This is what Sara McGlinchy tried to bring across. She tried to find a way to measure lung damage so that you know how much damage is suffered by sheep at 13,000 ft. What percent of the lung can be damaged without impairing the health of the individual? An animal at 5,000 ft. could probably get by on 1/3 of one lung. Human beings do this. At 13,000 ft. you have got to have more lung capacity than that. Every time you do anything to disrupt this lung capacity, whether it is lungworm or something else, this makes it incapable of surviving. In Alaska, where sheep are ranging at 2000 ft. they won't have the same problem as far as quantitative lung damage.

COMMENT BY TOM THORNE, G & F, WYOMING: I think, as George mentioned, they evolved together. I don't think we have any indication at all that lungworm is going to wipe out its host. I think it limits them and in some cases is pretty damaging, but if you consider all bighorn sheep I don't think it will ever wipe them out. I think it would be other factors much more important than lungworm that would wipe out the bighorn sheep.

Concerning stress, in Wyoming we have closely examined many sheep where we felt stress was directly involved and never have we incriminated or felt lungworms were important in a stress pneumonia. I am not trying to discredit lungworms. I feel they are important. The lungworms may help stress pneumonia to get started. The actual stress pneumonia does not require lungworms to kill sheep. It is generally a factor. *Pasteurella* seems to be most commonly involved, but who knows, there may be a virus or something else. The sheep die far too fast for a parasite. I have seen them show symptoms in the morning and die in the afternoon.

We have great difficulty in determining what is stress. Sheep on range look like the most calm animals in the world, especially some herds on winter ranges. You can walk around them and drive around them and they show very little concern. We can catch and put them in a trough and they don't look frightened at all. They look far more relaxed than any other wild animal which has been in captivity for a day or two, but I think inside, psychologically and physiologically, they are screwed up even though they don't show it. I don't think lungworms are involved in stress pneumonia.