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# SHEEP DISEASE STUDIES

by

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Volume I
Project Progress Report
Federal Aid in Wildlife Restoration
Project W-17-3, Job 6.6R (2nd half)
Project W-17-4, Job 6.6R (1st half)



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JOB PROGRESS REPORT (RESEARCH)

State: Alaska

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Job No.: 6.6R Job Title: Dall Sheep Diseases and Parasites

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SUMMARY

Necropsies of a series of Dall sheep collected on Crescent Mountain, Kenai Peninsula, from November, 1970 through April, 1971, revealed a variety of parasitic and/or chronic disease conditions.

Forty-one of 46 animals showed lesions caused by the lungworm *Protostrongylus stilesi* Dikmans, 1931, which was identified by the typical morphology of the spicules, gubernaculum and telamon seen in pieces of male worms recovered from the lesions. Several of the animals which did not show grossly evident lesions nevertheless were shedding lungworm larvae in their fecal pellets. Typical, whitish-colored lesions with surface nodulation ranged up to 35 percent of lung volume in relative size. Average lesion size was about 8 percent in both sexes. Lesions were seen in all age classes examined including lambs and two animals 13 and 14 years of age. Two ewes brought into town alive for experimental purposes from the Dry Creek study area near Fairbanks both succumbed to pneumonia within one to five months. The later death appeared to be a typical, chronic case involving lungworms and *Corynebacterium pyogenes* complicated by active "lump jaw" abscesses. The animal was otherwise in apparently prime condition with extensive deposits of fat.

A variety of trichostrongylid roundworms were recovered from the digestive tracts of the animals taken from the Crescent Mountain herd. Several other kinds of parasites including whipworms (*Trichuris* sp.) and coccidia (*Eimeria* sp.) were also encountered. Six fecal samples from each animal were quantitatively examined to determine the variability of numbers of eggs and larvae produced by the known helminth burdens. Considerable variability (i.e., 100 percent or more) was seen in individual infections. None of the helminthic or coccidial infections were considered to be more than low-grade judging by domestic animal standards.

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## BACKGROUND

Relatively little published information is available on the diseases and parasites of Dall sheep (*Ovis dalli*) in Alaska, or elsewhere. Goble and Murie (1942) reported the occurrence of a genus of lungworms, *Protostrongylus*, which is commonly associated with verminous pneumonia in bighorn sheep (*Ovis canadensis*) elsewhere. Murie (1944) recorded lump jaw as a common affliction of Dall sheep in McKinley Park. Philip (1937) noted the occurrence of the larvae of *Taenia hydatigena* (Pallas, 1776) in Alaska Dall sheep. Rausch (1951) failed to find any helminths in Dall sheep taken near Anaktuvuk Pass in the Brooks Range.

Studies on selected Dall sheep populations involving manipulation of numbers which have been recently initiated must take into account the possible effects of pathogens as well as weather, nutrition, etc. Because disease is known to be an important factor in the welfare of wild sheep populations elsewhere (Forrester, 1971) it seemed especially important that it be critically evaluated in our Alaskan Dall sheep population studies.

## OBJECTIVES

To qualitatively and quantitatively evaluate diseases and parasites as potential limitations to Dall sheep populations in the Kenai Peninsula.

## PROCEDURES

All sheep collected under Job 6.4 were subjected to a careful necropsy. Samples of presumed pathological conditions and parasites were preserved and analyzed in the laboratory.

In conjunction with Job 6.1 one or more sheep were trapped and brought into holding facilities in Fairbanks for study. Samples of fecal pellets were collected from each deposit during a 24-48 hour long period. These pellets were quantitatively analyzed using centrifugation to ascertain qualitative and quantitative parasite differences between deposits. Fresh fecal pellets were collected at each study area and qualitatively analyzed for parasite burdens. Mandibles from hunter-shot

and collected sheep were analyzed to determine the incidence of mandibular disease.

## FINDINGS

The present report is concerned with summarizing the work accomplished during the past segment on the parasites and diseases of Dall sheep. About fifty animals were collected by the sheep study leader on the Kenai Peninsula in groups of 5-10 on several occasions from November, 1970 to April, 1971. These were taken intact into the Fish and Game facilities at Soldotna and examined. Because trained and/or experienced personnel were not available, the results of the necropsies may not be entirely representative of the state of health of the animals. However, the respiratory and digestive tracts and associated organs were removed intact and sent to the Wildlife Disease Laboratory in Fairbanks for further examination. These results are reported below.

Additional studies on parasites and diseases of Dall sheep were carried out on the Dry Creek population. Serum was taken for disease testing from about 100 animals during trapping and tagging operations. Two animals were brought in at the end of trapping operations and held for further studies on "lumpy jaw" and lungworm.

Observations were made on the prevalence and related conditions of several groups of pathogens: Lungworm, gastro-intestinal parasites (including coccidia), and lump jaw organisms. These are all separately considered below.

### A. Lungworm.

#### 1. Identification

Until the present time no one has reported the identity of the species of worms that infect the lungs of Dall sheep, in Alaska or elsewhere. Goble and Murie (1942) suggested that fragments of worms seen in sections of Dall sheep lungs were probably of *Protostrongylus* but they did not examine adult specimens closely enough to establish that they were, in fact, referable to the genus they cited. The Alaskan record for *Protostrongylus* in Dall sheep by Forrester (1971) is based on the uncritical acceptance of Goble and Murie's (1942) essentially unsupported assumption and is, therefore, without scientific merit.

Recently we were able to obtain useful fragments of male specimens from an animal from Crescent Mountain, Kenai Peninsula. These specimens clearly showed the anatomical details of the spicules, gubernaculum and telamon which unequivocally identify *Protostrongylus stilesi* Dikmans, 1931.

In 1963 we found typical, lateral-thorned larvae of *Muellerius* sp. in one sample of fecal pellets from the Wrangell Mountains. Subsequently, a specimen of lung tissue from a ram killed by a hunter in the Chugach

Mountains was submitted to Dr. Jack King, Agriculture Research Service, U.S.D.A. VIC-Alaska. He sent the specimen to a U.S.D.A. diagnostic laboratory and was informed that it was infected by the larvae of *Muellerius minutissimus* (Mognin, 1878), a species not known from wild sheep in North America at that time. More recently larvae of *Muellerius* have been commonly seen in fecal pellets of California bighorn sheep in British Columbia (Bandy, personal communication).

We have failed to find other examples of *Muellerius* larvae in about fifty sets of lungs from Kenai animals and several hundred sets of pellets from the Kenai Peninsula, Wrangell Mountains, Alaska Range or Brooks Range.

In December, 1963, a dead lamb was found on the Dry Creek study area. It had four, large strongyline nematodes in the bronchioles of its lungs. Although the specimens were evidently lost during the Great Anchorage Earthquake, it seems likely that a tentative, though unverifiable, diagnosis of *Protostrongylus rushi* Dikmans, 1937, is warranted. No other lungworms referable to this species have been seen in Alaskan Dall sheep.

2. Lungworm Lesions

*Protostrongylus stilesi* is a tissue parasite, unlike the bronchiole-dwelling *Dictyocaulus viviparus*, a common parasite of other Alaskan cervids, or *P. rushi*. The hair-like adults of *P. stilesi* intertwine amongst the cellular and connective tissue components of the lung tissue and are very difficult to recover for study. Their presence in intact l lungs is indicated by nodules and/or distinctly whitish, emphysematous areas principally around the posterior margins of the lungs.

It has been the practice for other students of lungworm disease in North American wild sheep to view such lesions as essentially two-dimensional structures (Pillmore, 1961; Forrester and Senger, 1964; Forrester, 1971). Thus the extent of lesions observed are often given in square millimeters of lung surface area. The fact is that two-dimensional measurements of the surface area of verminous lesions are a poor estimate of the actual size or extent of lesions which indeed are actually three-dimensional structures.

Accordingly, we concluded at the start of our study that we must attempt to measure the volume of affected tissue if we were to accurately estimate the relative extent of lesions in different animals. The procedure we devised is relatively simple. First, the total volume of the lung is measured by displacement. Next, the lungs are thinly sliced in a slightly frozen condition. The infected tissue is relatively whitish in contrast to the normal, red lung tissue. The affected parts are trimmed out and their volume measured by displacment. The percentage of each lung thus infected is easily calculated. To confirm that so-called affected areas were indeed infected with lungworm larvae, five small, randomly selected samples of tissue from each infected pair of lungs were crushed on a slide and examined for "spike-tailed" larvae

(*Protostrongylus*) or "lateral-thorn-tailed" larvae (*Muellerius*). Over 2,000 larvae were examined but only those of *Protostrongylus* were seen. Pieces of adult *Protostrongylus* were only occasionally encountered.

One can heighten the contrast between infected and normal tissue by first fixing the intact lungs in a 5 percent formalin solution. The lungs are then also easier to slice following fixation but are not as satisfactory for recovery of pieces of adult worms.

McGlinchy (1971) presented a procedure for more accurately measuring lungworm infestations in wild sheep than had been reported previously. The method involved a combination of counting larvae in histological sections and "Baermannizing" the remainder of the lung tissue. The weights of nodular and non-nodular tissue were also recorded. In this manner, no doubt, a reliable estimate of the total number of larvae infecting a set of lungs and the percent by weight of the lesions can be made. However, the total number of larvae may be only indirectly related to actual lesion size and character, and the weight of lesions is less directly related to physiological function than volume. In addition to size, the potential significance of lungworm lesions may be related to two other characteristics. The emphysematous character of a lesion is probably the principal determinant of the chronic course of the infection. The susceptibility of a lesion to secondary invasion by bacteria (e.g., *Corynebacterium pyogenes*) which cause pneumonia will determine in part the likelihood of acute disease. Whether or not these two characteristics are separately or identically related, directly or otherwise, to numbers of larvae per unit of lung tissue is unknown.

Experimental infections reported by McGlinchy (1971) suggest that there is not a direct correspondence between total first-stage larval burdens and percentages of infected (i.e., nodular) tissue. This worker fed identical numbers of presumably infective larvae to two bighorn/mouflon hybrids. At sacrifice one animal harbored seven times as many adult worms (14 vs. 2) and three times as many first-stage larvae (34,000 vs. 7,000). However, the ratios of histologically or grossly "disrupted" to normal lung tissue were reversed. That is the more heavily infected animal (adult and larval worms) had smaller percentages of "disrupted" lung tissue (i.e., 0.8 and 0.9 vs. 4.2 and 2.7, respectively). One can only suppose that these results are typical of small-scale experimentation. I am inclined to intuitively suppose that the less heavily (fewer first-stage larvae) but more extensively (greater percentage of lung volume) infected animal would be more emphysematous while the other animal might be somewhat more prone to secondary bacterial invasion and consequent pneumonia. However, it may be that an emphysematous response requires only a similar or even a higher larval density than does susceptibility to secondary infection. Until these distinctions (if indeed they are valid) are resolved, it seems to me that the potential chronic effects of lungworm lesions are best estimated by simply measuring by volume the relative sizes of the lesions encountered. The older surface area measurement appears to be of relatively lesser value in this regard.

The prevalence and relative size of lungworm lesions are shown in Table 1 and Figs. 1 and 2. While the data are not adequate to describe

Table 1. Prevalence and relative size of lungworm lesions in lungs of sheep from the Crescent Mountain study area.

| Sex | Age (yrs) classes | Sample Size | Relative size of lesions: Range Infected | Range Negatives | Average per number infected |
|---|---|---|---|---|---|
| male | 1 | 5 | 3.9-19.0 | none | 11.7 |
| male | 1 | 4 | 1.5-12.6 | none | 7.8 |
| male | 2 | 4 | 0.2-9.0 | 1 | 4.9 |
| male | 3 | 1 | - | none | 4.0 |
| | All | 14 | 0.2-19.0 | 1 | 8.3 |
| female | 1 | 2 | 2.2-11.0 | none | 6.6 |
| female | 1 | 5 | 3.9-14.0 | 1 | 5.9 |
| female | 2 | 4 | 5.3-17.0 | none | 10.3 |
| female | 3 | 4 | 0.2-6.6 | none | 2.8 |
| female | 4 | none | - | - | - |
| female | 5 | 4 | 0.9-11.0 | 1 | 4.5 |
| female | 6 | 4 | 4.8-11.5 | 1 | 7.8 |
| female | 7 | 2 | 3.9-21.0 | none | 12.5 |
| female | 8 | 2 | - | 1 | 0.7 |
| female | 9 | 2 | 0.3-17.3 | none | 8.8 |
| female | 10 | 1 | - | none | 15.0 |
| female | 11 | none | - | - | - |
| female | 12 | none | - | - | - |
| female | 13 | 1 | - | none | 8.2 |
| female | 14 | 1 | - | none | 35.4 |
| | All | 32 | 0.2-35.4 | 4 | 7.3 |

FIG 1. Intensity of lungworm infections in Crescent Mtn. Dall sheep.

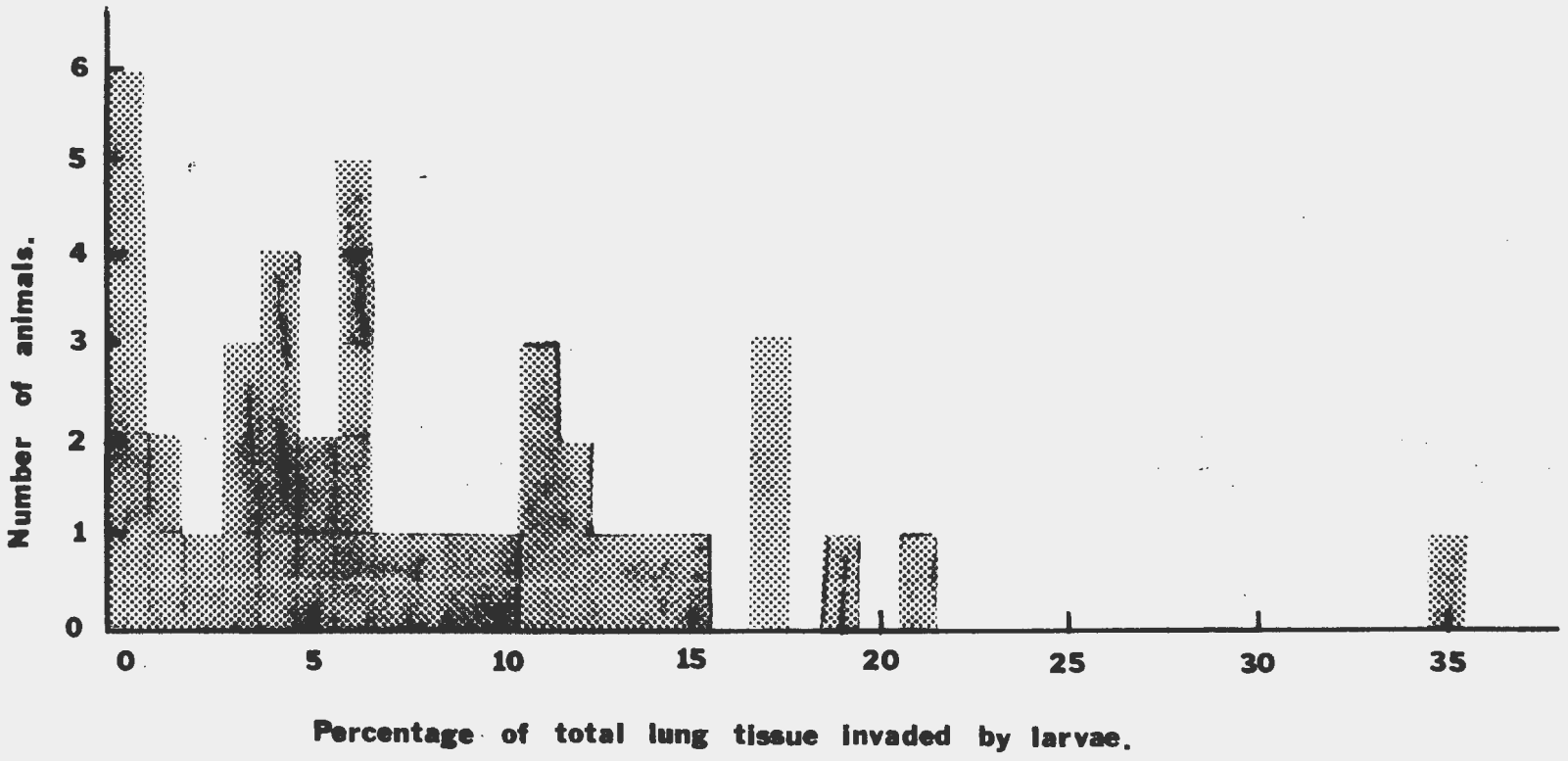
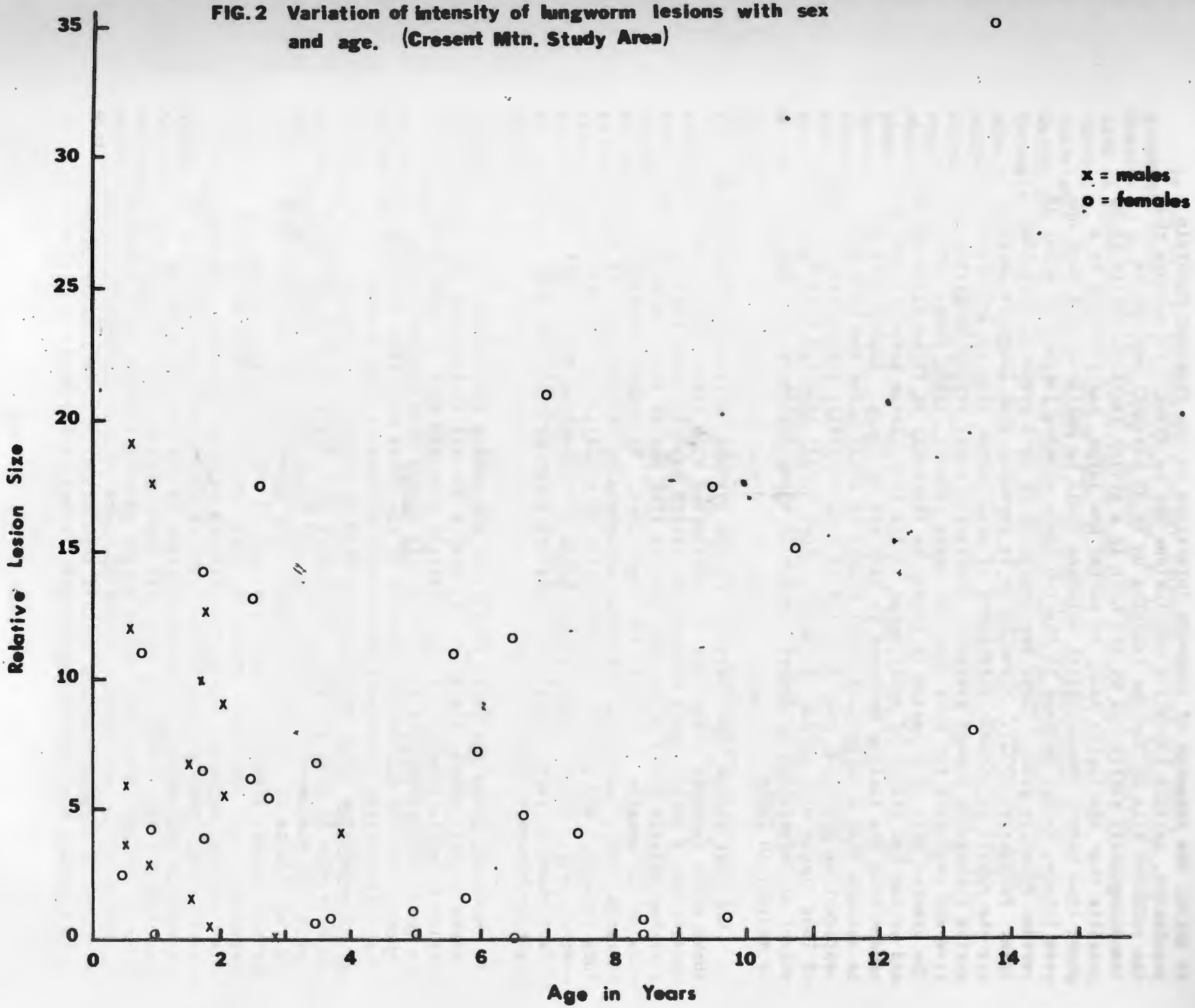


FIG. 2 Variation of intensity of lungworm lesions with sex and age. (Cresent Mtn. Study Area)



in detail the dynamics of lungworm infections in the Crescent Mountain population, certain tentative conclusions may be drawn. It appears that the relative size of lesions (ranging up to 35 percent) may not be immunologically restricted by the host in a strict fashion. While it is possible that any lesion seen, no matter how large, was produced in a short time before immunological defenses could be mustered this does not seem likely, except perhaps in the two lambs showing lesions of about 18 percent relative size. These may represent in part prenatal infection (see: Forrester, 1971). Normally it is expected that lungworm lesions are the result of chronic infections of adult worms plus re-infections during subsequent summer seasons. Apparently adult protostrongylids may live and reproduce for more than two years (Kassai, 1962; Dunn, 1969). The greater part of a lesion is caused by thousands of first-stage larvae, and we saw few pieces of adult worms in the five tissue samples taken at random from the lesions observed in each set of lungs. Accordingly it is assumed that a relatively small number of adult worms may have the reproductive potential over a period of time to infect substantial volumes of lung tissue with hordes of larvae. Boev (1957) reported that the extent of infestation in domestic and wild sheep increases with age and is minimal in lambs.

One must assume that there is, on the average, a critical-lesion-size above which lungworm infections in combination with other factors may consititute a particularly significant health hazard. More heavily infected animals would be at some greater risk and at any time would have on the average a shorter life expectancy. Secondary invasion by bacteria, etc., leading to verminous pneumonia or reduced vigor (i.e., lung capacity) and greater susceptibility to predation or adverse weather, etc., are but two possible kinds of mechanisms which might affect sheep welfare. Critically infected males probably are less vigorous breeders (Geist, 1971).

However, Cowan and Geist (1971), in discussing the effects of disease on wild sheep, state (p. 76) that a die-off of bighorns in British Columbia involved "sheep...heavily infected with a lungworm of the genus *Protostrongylus* that has <u>little effect</u> upon well-nourished bighorn." In the following paragraph on the other hand they conclude that, "A bacterial eruption (*Pasteurella*) occurred in the already <u>parasite-weakened</u> lungs and the sheep died of pneumonia" (special emphasis mine in both quotes). It appears that there is some contradiction in the statements by Geist (1971) and Cowan and Geist (1971) regarding the effects of lungworm on sheep. They appear to confuse acute with chronic disease effects in their writing, if not otherwise. Acute responses (e.g., secondary pneumonia, fatal or not) probably occur most often only in conjunction with malnutrition or other stresses. Chronic effects (e.g.. reduced lung function and related vigor) are likely principally related to the relative volume of the lung(s) which is deactivated. Dunn (1969) reports that experimental infections of lungworms in domestic sheep may reduce weight gain by as much as 17.7 percent. It remains to be seen whether or not adequate data on the prevalence and intensity of lungworm lesions in wild populations will eventually allow us to estimate the risk associated with chronic infections.

It may be that increasingly larger lungworm lesions are not a necessary consequence of increasing age, at least on the Kenai Peninsula. Whether or not this is solely a matter of individual host resistance, lack of opportunity for infection or possibly a combination of both factors is not known. Perhaps there are cyclic variations in herd resistance and external conditions favoring the lungworm life cycle which, occurring together or independently, may determine prevalence and intensity of lungworms in Dall sheep at any particular time. Nutrition also no doubt has some influence in these matters, particularly on the occurrence of acute sequelae, i.e., verminous pneumonia. In areas where climatic and other factors favor opportunity for infection, there may be considerable natural selection for resistant animals. In other areas where natural conditions are generally unfavorable for the extra-host phase of the lungworm cycle, herd resistance may be maintained at lower levels. Under these circumstances occasional periods of particularly favorable conditions might lead to periodic increases in opportunity for infection (i.e., epizootics). If increased opportunity for infection coincided with high host population density, under-nutrition and severe winter conditions the stage would be set for an episode of verminous pneumonia. In the absence of nutritional and/or climatic stresses, the outcome would tend to be of a chronic nature. In areas where opportunity for infection was highly variable, one would expect to see a greater age-relatedness of lesion prevalence and intensity. That is, particular year classes would show higher average lesion size. The degree to which lungworm larvae are able to survive winter conditions and larval longevity on various Alaskan sheep ranges are unknown. Therefore, it is difficult to convincingly estimate the likelihood of carry-over and buildup of larval lungworm populations in our study areas. However, Forrester and Senger (1963) conducted controlled laboratory experiments which led them to conclude that temperature and humidity would not significantly influence the survival of first-stage larvae of *Protostrongylus stilesi* in fecal material in Montana. Alaskan strains may be as well adapted to climatic extremes. Survival and reproduction by adult worms of up to 28 months duration (Kassai, 1962) by the domestic sheep protostrongylid, *P. rufescens*, allow for some degree of unluckiness with seasonal environmental variation on the part of larvae and the snails in which they further develop.

Much additional field data, but also experimental studies, are required to adequately understand the epizootiology of lungworm in Dall sheep and the effects of the parasite on host populations.

### 3. Pellet Studies

Because it is difficult or otherwise impractical to collect adequate numbers of Dall sheep for parasitological studies, we have been exploring for some time now the use of fecal pellet analysis for eggs or larvae of parasites as a means of investigating the problem. While there is little argument over the qualitative value of data derived by means of pellet analysis, the quantitative value of this kind of data seems less certain.

The methods used for pellet analysis are designed to concentrate larvae or eggs from a given amount of feces in order that they may be easily observed and/or counted. The principal method that has been used to enumerate lungworm larvae in fecal pellets is the so-called Baermann technique as described in Forrester (1971). This procedure depends upon the normal thermotropic behavior and vigor of lungworm larvae and will not work with dead larvae. Various flotation or sedimentation methods which employ suspending fluids of carefully selected specific gravities (e.g., saturated zinc sulfate, Sheather's sugar solution, etc.) depend only on the normal specific gravity of the eggs or larvae under study. Which kind of procedure yields the most accurate quantitative data with the material we have at hand is still under study. The McMaster's flotation procedure which we use employs the "Fecal Counting Chamber Kit" #H-L 4100 produced by Haver-Lockhart Laboratories. The McMaster's procedure is much more convenient to use than the more time-consuming Baermann technique. It has the added, great advantage that it provides data not only on lungworm larvae, but also nematode eggs and other larvae and coccidial oocysts. Whether one or the other technique has greater absolute accuracy may ultimately be of small interest since relatively accurate results would be sufficient for most of our purposes.

The data on the analyses of pellets from animals from the Kenai and Dry Creek study areas are considered together in the following sections.

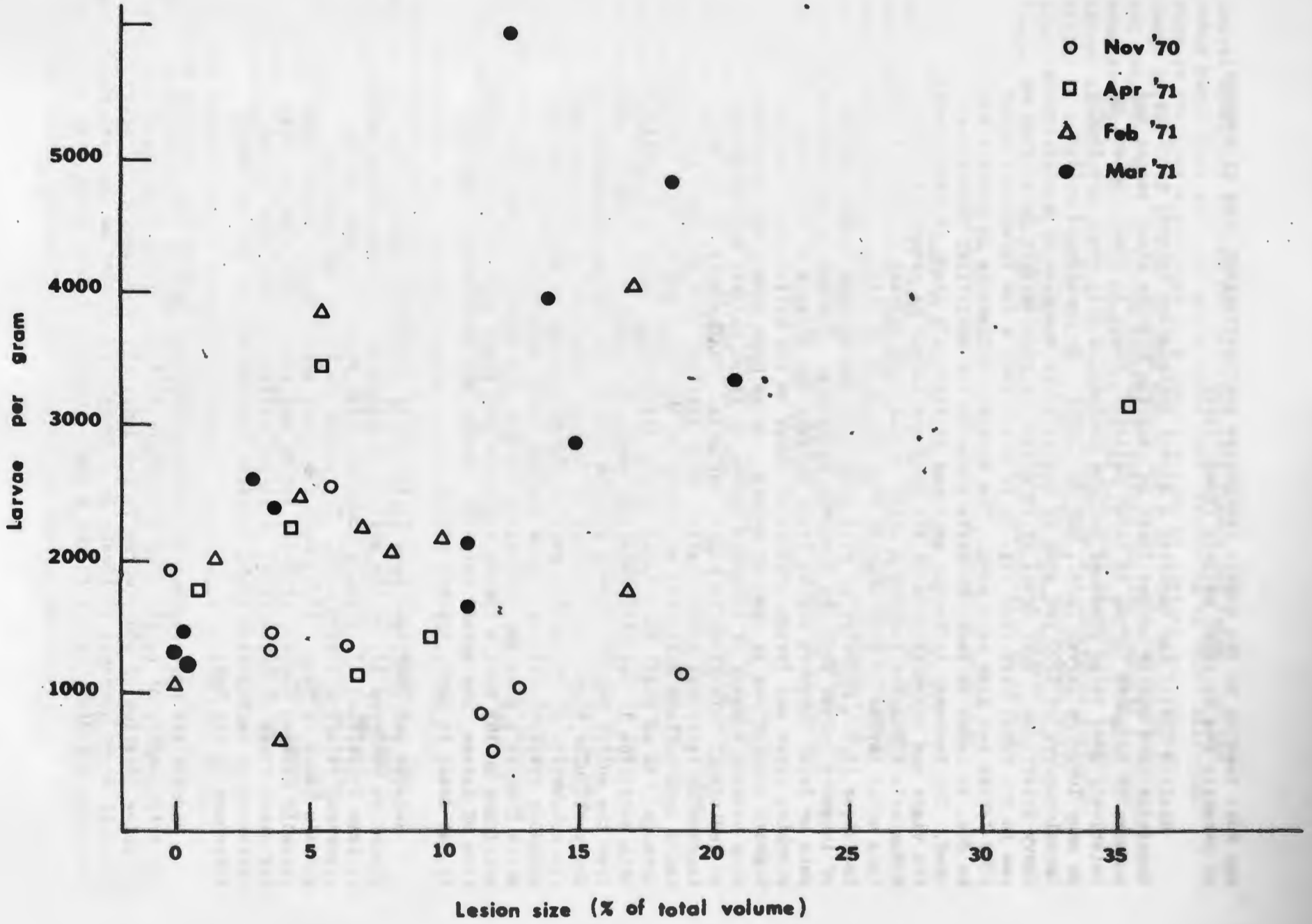
a. Lesion Size and Larval Numbers

The analysis of fecal pellets was designed with several purposes in mind. I wanted to determine what kind of correlation might exist between the number of lungworm larvae in pellets and the size of associated lungworm lesions. I also wanted to determine the variability of larval parasites, etc., from one time of day (i.e., fecal deposit) to the next. This latter purpose is considered in the next section.

The relationship between the volume of lesion tissue that is grossly visible and the average number of lungworm larvae per gram of fecal pellets (six samples per individual) is shown in Fig. 3. It is apparent that substantial numbers of larvae are being released before the affected sites in the lungs are readily visible to the naked eye. If we assume that the average number of larvae per unit of feces is directly related to the average density of larvae in lesions, then it appears that some lesions are more intensely infected than others. This is precisely what was observed in the two experimental infections reported by McGlinchy (1971) that were considered earlier in this report.

There are other factors which probably complicate the lesion-pellet-larvae relationship. Larger (and older ?) lesions may not be as productive of larvae as smaller (and younger ?) lesions. To what extent host immune responses affect larval production and release is also uncertain. However, in domestic sheep it is clear that host immunological defenses can delay maturation and egg production of trichostrongylids. These reproductive activities are released from immunological inhibition during the stresses of parturition and the early post-parturient state of

FIG 3. The relationship between gross lesion size and numbers of lungworm larvae in fecal pellets (Kenai Study Area).



the host results in the widely recognized post-parturient rise of numbers of parasite ova in fecal pellets (Dunn, 1969).

Stelfox (1971) has reported a direct correlation between stocking densities (and winter weight loss) on Canadian bighorn sheep ranges and numbers of lungworm larvae in fecal pellets. Sheep in lower numbers on relatively good range produced pellets containing only about one-fourth as many lungworm larvae (i.e., 600 vs. 2400). He considered pellet-larval-numbers of 1200 per gram or higher to be examples of significantly heavy infections. This level of infection is considerably lower than we saw in the Kenai herd (see Fig. 3) in which only a few infections under 1200 larvae per gram were seen. The apparent difference in numbers may be due, at least in part, to differences in the analytical techniques used. The Baermann technique employed in Stelfox's study is based upon the vigor and thermotropism of the lungworm larvae he recovered. The McMaster's flotation technique which we use will recover lungworm larvae (and other larvae or ova) whether they are dead or alive. I assume our technique (i.e., McMaster's) will routinely demonstrate higher numbers of lungworm larvae than the Baermann procedure. Whether or not Stelfox's data on fecal lungworm larvae truly reflect the effects of different stocking rates and forage production rates on the well-being of several bighorn populations is open to debate. As we have shown, the relationship between numbers of lungworm larvae in fecal pellets and intensity of individual infections is highly variable. Furthermore, it is my impression (personal communications with Stelfox) that his data are based upon pellets collected at random on the range and not from specific animals. As we shall show later in our discussion, there is a considerable variation in parasite densities among different fecal samples taken from the entire mass of formed pellets present in individual animals at any particular time. Our "larvae to lesion" relationship is based upon six subsamples of the entire mass of formed pellets in each animal and probably realistically estimates average fecal densities of larvae. While Stelfox's data may accurately reflect the effects of variable nutrition on the host's immune responses and their inhibition of production of larvae, the actual severity, extent and prevalence of lesions is, at least in part, another matter.

Because our specimens were collected at different times of the winter we wondered if, as the animals declined in condition, there would be less inhibition of larval production which would then more nearly represent relative lesion size. The points in Fig. 3 representing animals taken in March, 1971, seem to better approximate a linear relationship between numbers of fecal larvae and lesion size than do those for other times of collection. A larger collection made late in the winter or at parturition might minimize the variability due to immune responses of the host.

Perhaps the best explanation for the apparent lack of linearity in the relationship of lesion size to numbers of fecal larvae involves the relative location of adult worms in the lung. If, for example, 10 pairs of adult worms happened to locate in a restricted cluster in one part of the lung, one might expect to see a small lesion with relatively high

larval densities in both the affected lung tissue and feces. On the other hand, five pairs of adult worms less restricted in their distribution in the lung might lead to a larger volume of lesions even though fewer larvae were present in lung tissue and feces. The histopathological responses of lung tissue leading to visible lesions probably require some average, "threshold-number" of larvae per unit of tissue. Higher larval densities might lead to further changes (e.g., increased susceptibility to infection) which are not grossly visible. Other than their predilection for the peripheral, principally posterior, margins of the lungs, it appears that adult lungworms locate essentially at random.

b. Individual Variability of Fecal Parasite Densities

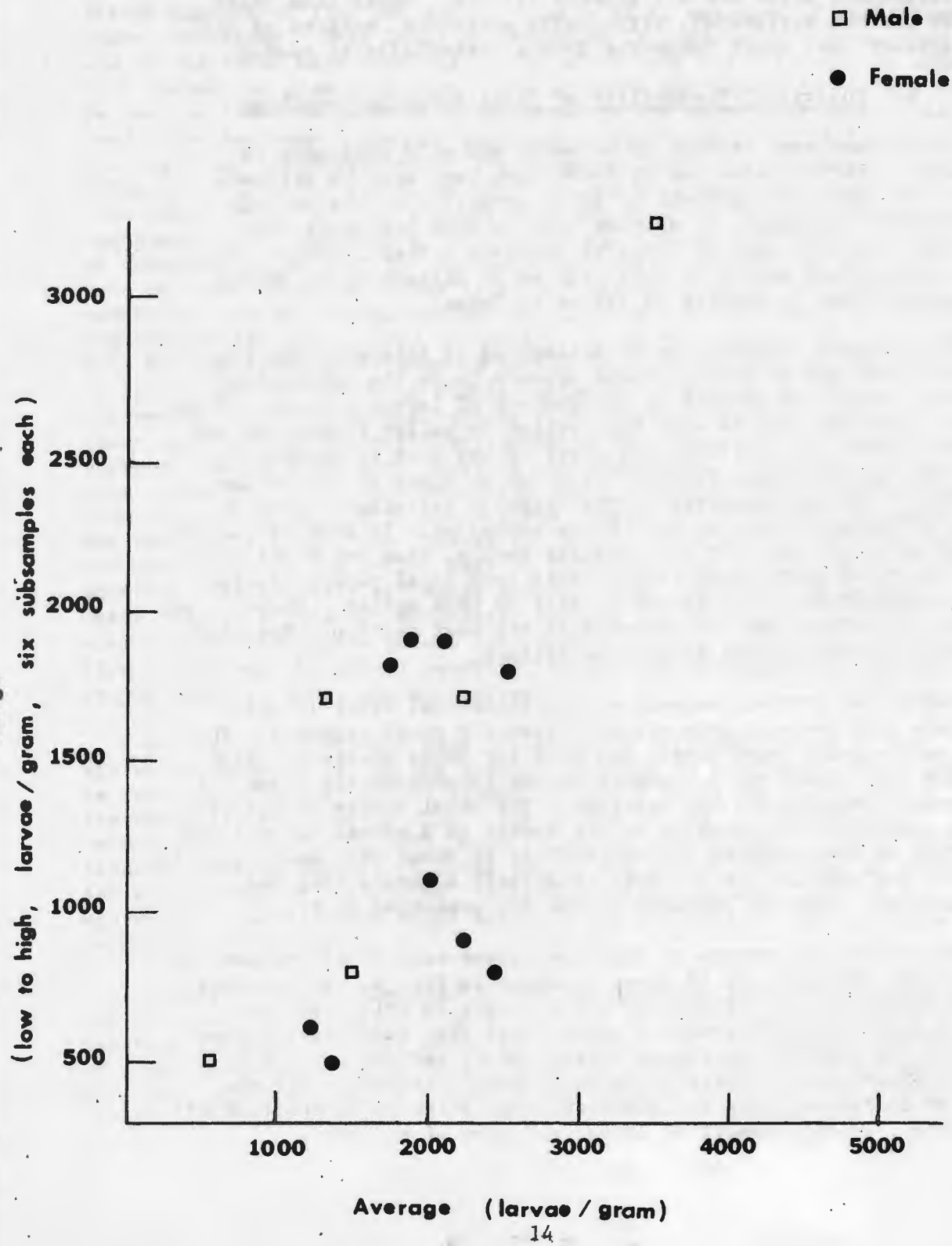
There have been many studies on domestic and wild ruminants in which numbers of larval parasites in feces have been used to estimate relative, if not absolute, numbers of adult parasites in the animals under observation. However, it appears that in many instances such estimates are in either case of doubtful accuracy (Gibson, 1965). We have just examined one aspect of this problem in attempting to relate lungworm lesion sizes to numbers of larvae in feces.

One of the major difficulties in attempting to interpret the significance of fecal egg or larval counts appears to be the variability from one fecal sample to another in the numbers of larvae present. These numbers have been reported to vary from pellet to pellet (Forrester and Senger, 1964), from one physiological state of the host to another (e.g., parturient vs. barren; Dunn, 1969) or from day to night or day to day (Gibson, 1965). We have wondered whether similar variations occur in the release of larval stages of Dall sheep parasites. If such variations as did occur were of a more or less regular nature, then one might still be able to retrieve useful quantitative data from fecal pellet studies. We used two approaches in our recent studies on this matter. Observations on a captive Dall sheep ewe are reported in the next section. Material from the Kenai collection was treated as follows.

We divided the terminal segment of the intestinal tract in which formed pellets were present into six approximately equal segments. The pellets in each segment were frozen and held for later analysis. Each subsample was then mixed and a standard volume (approximately 2 gms. of feces of normal consistency) was analyzed. The usual number of pellets per segment appeared to be similar to the number in a normal deposit and I suppose that we have sampled the variability in about that many deposits. I expect that our results are at least relatively accurate from one animal to another. Data on lungworm larvae are presented in Fig. 4.

The variability in numbers of lungworm larvae seen in six subsamples of fecal pellets from each of 13 sheep is shown in Fig. 4. If our data are representative of the variability which occurs in Dall sheep and other host-lungworm combinations, it seems clear that quantitative observations which are reported on single subsamples of pellets are likely to be relatively inaccurate. There seems to be some correlation between the amount of individual host variation seen and relative intensities of infection (i.e., average number of larvae per gram of pellets).

FIG 4. Variation in numbers of lungworm larvae in fecal pellets from individual infections.



c. Diurnal Variation of Numbers of Lungworm Larvae in Fecal Pellets

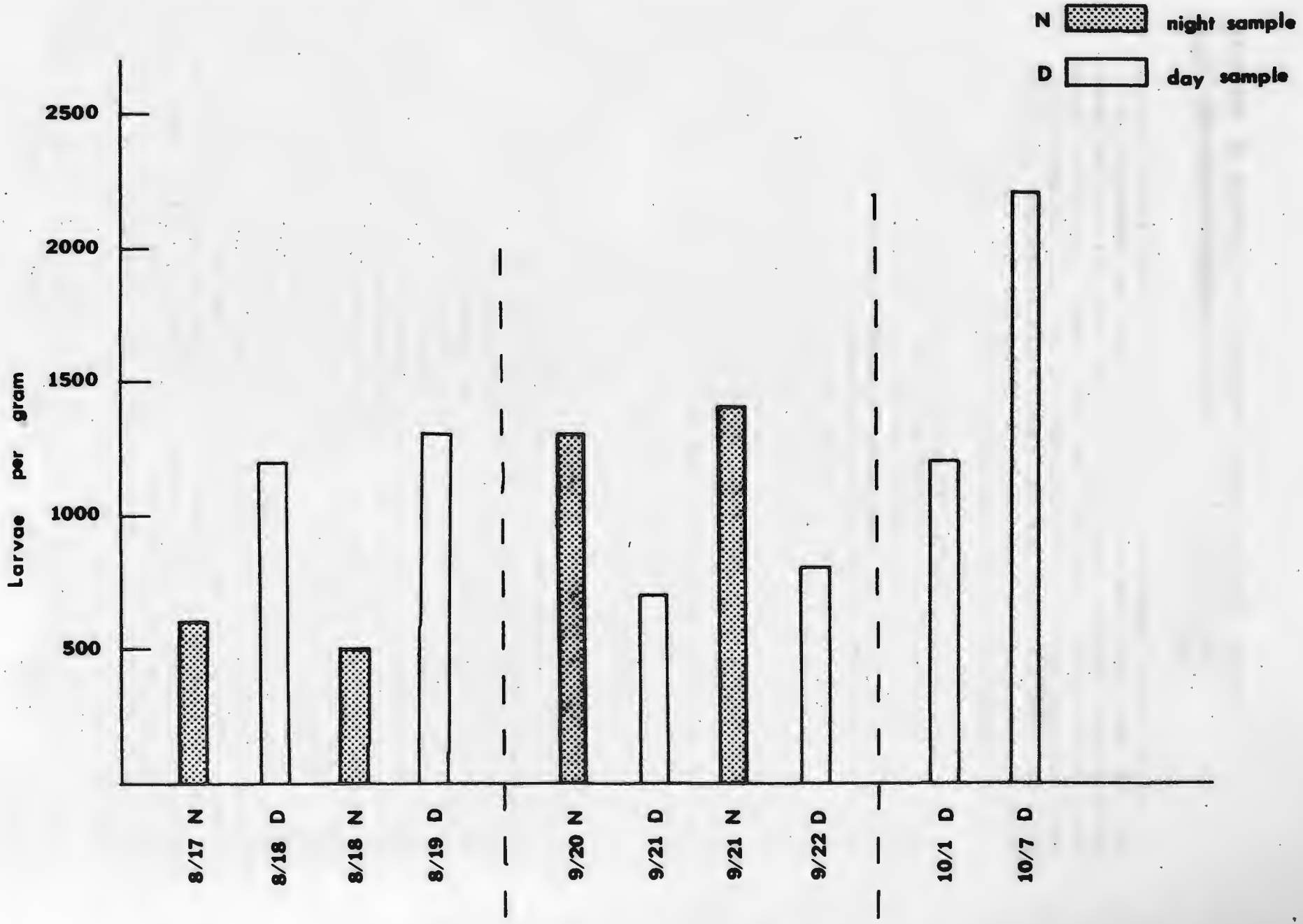
At the end of tagging operations at the Dry Creek lick in June, 1971, two adult Dall ewes were brought live into the laboratory for extended parasitological studies. Within two weeks one had died from a "shipping fever-like" condition. The other animal, a veritable, living parasitological garden, lived on in robust health until December when she, too, rapidly sickened and died from a massive verminous pneumonia. Further comments will be made about the etiology of this case in the following section and a later one dealing with lump jaw studies.

In order to study diurnal variations in the release of lungworm larvae in fecal pellets we collected all of the pellets dropped from 8:00 a.m. to 8:00 p.m. (day sample) and 8:00 p.m. to 8:00 a.m. (night sample). Each day or night sample was thoroughly mixed and five or six subsamples were taken for analysis by the McMaster's procedure. The results are shown in Fig. 5. It appears that diurnal variations may occur but more observations are needed to determine what the normal regularity may be. According to Gibson (1965), Spedding has reported (in a paper not available to us) "considerable variation" in samples taken at different daily intervals. Spedding suggested that, when attempts were made to detect slight differences in infection, counts should be made on a sample drawn from the well-mixed daily fecal output of the animal at question.

4. Verminous Pneumonia

This complex disease is recognized as a major contributing factor to precipitous declines in bighorn sheep populations in Canada and the United States (Forrester, 1971). Although we have had several first- or second-hand reports over the years of Alaskan Dall sheep showing signs of respiratory distress, we know of no bona fide case of this condition in any Dall sheep population. However, the lungworm, *Protostrongylus stilesi*, which has been implicated in this disease syndrome is present on all of our sheep ranges. In some instances (i.e., Kenai and Dry Creek study areas), it appears that these Alaskan lungworm infections are comparable to those which are judged to be significant in bighorns. We do have two clear-cut cases of verminous pneumonia in captive Dall sheep. Both animals (adult ewes) were brought into Fairbanks from the Dry Creek study area in late June, 1971. They were kept in a pen which had previously been used to hold pigs at the Arctic Health Research Center animal facility at Ft. Wainwright. On August 2, both animals appeared normal but the following day #742 (sheep lick tagging system) appeared lethargic and was treated by Dr. Maury Hamlet, Post Veterinarian. It was treated again the following morning about 8:00 a.m. and was found dead one hour later. A brief summary of the results of the necropsy performed with the help of Dr. Hamlet and histopathology by Dr. Rollo Van Pelt, Institute of Arctic Biology, University of Alaska, is as follows.

FIG 5. Diurnal variation in numbers of lungworm larvae in fecal pellets.



#742, female, 74 months old

The animal was generally in good condition with some fat on the internal organs. Although the molariform and incisiform teeth were somewhat loose there were no signs of lump jaw. The primary internal lesions involved the lungs and kidneys.

Numerous lungworm larvae were seen in tissues submitted for histopathology and about 1.4 percent of the total volume of lung tissue was macroscopically involved in typical lungworm lesions. Many of the alveolar spaces were filled with neutrophils and numerous bacterial bodies superficially resembling *Escherichia coli* in smears. Some alveoli contained necrotic material.

Sections of kidney tissues revealed tubular atrophy throughout the specimens examined.

Parasitological examinations revealed about 90 trichostrongylids (probably *Ostertagia* sp.) in the abomasum. Other parts of the digestive tract were free of worms. *Sarcocystis* sp. was numerous in skeletal muscle samples.

The clinical diagnosis by Dr. Hamlet was listed as "pneumonia." The histopathological diagnosis by Dr. Van Pelt was: "Subacute bronchopneumonia compounded by the presence of lungworms, renal tubular atrophy and a generalized lymphocytic depletion involving the lymph nodes."

The other ewe (#690), about ninety months old at death on December 22, 1971, was in apparent robust health until about one week prior to her demise. Our earlier observations revealed that she was a veritable parasitological museum. There were simultaneously, active infections of gastro-intestinal trichostrongylids, lungworms, coccidia and well developed lump jaw lesions. These will be discussed further in a later section of the report.

Several weeks prior to the death of #690, it was necessary to move her to new quarters in the Arctic Health Research Center's animal compound on the campus of the University of Alaska. Part of a pen used to hold domestic sheep was partitioned off and a three-sided shelter was built. The animal seemed to adjust to the new surroundings but displayed some degree of lethargy about one week before death. A brief summary of the results of the necropsy performed with the cooperation of Dr. Robert Dieterich and Dr. Rollo Van Pelt, Institute of Arctic Biology, University of Alaska, is presented below.

#690, female, 90 months old

The animal was in very good condition with substantial stores of internal and subcutaneous fat. Both mandibles were well involved with extensive lump jaw lesions from which two agents were subsequently isolated (i.e., *Corynebacterium pyogenes* and *Fusobacterium necrophorum* - *Spherophorus necrophorus*). A more detailed discussion of this case of lump jaw will be presented in a later section of this report.

The organs of the thoracic and abdominal cavities showed numerous lesions. The lungs were partially to completely consolidated with numerous abscesses throughout their parenchyma. A pronounced pericarditis was evidenced by a marked thickening of the pericardial sac and a pronounced fibrino-purulent reaction involving the epicardium. This response resulted in numerous adhesions between the pericardial sac and the epicardium. There were adhesions between the serosal surfaces of both lungs and the wall of the thoracic cavity. Numerous abscesses were present in both kidneys. The adrenals were inflamed.

Parasitological examinations revealed moderate infections of several species of both gastrointestinal, trichostrongylid nematodes and the coccidian genus *Eimeria*. Lungworm larvae were present in relatively large numbers in fecal pellets and in some sections of lung tissue.

Pure cultures of *Corynebacterium pyogenes* were recovered from specimens of lung and kidney tissue.

The cause of death was diagnosed as involving chronic, suppurative bronchopneumonia, fibrinopurulent pericarditis, centrolobular degeneration of the liver, suppurative nephritis and inflammation of the adrenals; all the result of a disseminated infection of *Corynebacterium pyogenes* complicated by a chronic lungworm infection. Well advanced, chronic "lump jaw" lesions also infected by *C. pyogenes* as well as *F. necrophorum* may have been the source of bacterial cells which led to the widely disseminated, internal infection.

According to Post (1971) *C. pyogenes* is one of the several bacterial species which have been commonly isolated from chronic cases of pneumonia in wild sheep. He considers simple, chronic lungworm infection to be another of the three kinds of pneumonia seen in wild sheep. A third form of pneumonia involves a combination of stress and one or another species of *Pasteurella* which are otherwise present under normal conditions in the respiratory tract of sheep. This latter type of pneumonia normally leads to acute septicemia and is rapidly fatal. Post also claims that when adult bighorns are brought into captivity they almost invariably develop some form of pneumonia and can only be saved through suitable antibiotic treatment. No doubt the stresses of captivity will sooner or later aggravate infections which might otherwise follow a more moderate course.

The amount of lung tissue involved in lungworm lesions in case #742 was small (i.e., about 1.4 percent) and we noted no signs of respiratory distress during the month prior to its death. The rapidity with which the animal visibly sickened and died suggests acute Pasteurellosis as described by Post. Unfortunately, the tissues taken for bacteriological assay by Dr. Hamlet were not cultured. Since there is some similarity i in the size and shape of *Pasteurella* to *Escherichia*, perhaps the "*Escherichia*-like" organisms seen in smears (see summary of case given earlier) were in fact the former.

Case #690 appears to be a typical chronic pneumonia involving a synergistic combination of protostrongyline lungworms and a bacterial agent (i.e., *C. pyogenes*) commonly seen in suppurative pneumonia. Whether or not the lump jaw lesions played an important role in the secondary infection of the lungworm lesions by *C. pyogenes* is not known. The jaw lesions were probably of longer standing than those in the lungs, but the earlier isolates from abscess material from the jaws included only two species of *Streptococcus* (i.e., *S. faecium* and *S. sanguis*). It would be interesting to know the composition of the jaw-lesion fauna several weeks or so before the animal died.

Finally it should be noted that the timing of the obvious sickening and death of #690 might be related to the move to the new quarters. Perhaps the stress of new surroundings and exposure through fencing to domestic sheep and their flora and fauna led to the acute conclusion of a chronic condition.

It seems clear enough that all the components of the verminous-pneumonia complex are present in Dall sheep. We also have various other contributing factors (e.g., notably long, hard winters and lump jaw) which no doubt can aggravate the condition. I expect that when our Dall sheep populations are known as well as are many bighorn populations in Canada and the western United States we will recognize occasional acute epizootics of the lungworm-pneumonia complex in Alaska. However, all to often one only sees the <u>results</u> of acute disease in wild populations, i.e., declines in numbers of animals. Retrospective studies which attempt to understand population declines by collecting animals after the fact, e.g., the Suprise Mountain sheep decline between June 1968 and March 1970, only tell you about the animals that survived. Thus if only slight indications of disease conditions or pathogens are seen in survivors, one has no assurance whatever that the animals that did die were also only "slightly diseased." Indeed, one must suppose that diseased animals would be among the first to succumb. While this concept seems simple enough, it is remarkable how often wildlife scientists draw unwarranted conclusions about past events from purely after-the-fact studies. Considering that we apparently don't even know when the Surprise Mountain decline occurred (i.e., the winter of 1968-69 or 1969-70) it is difficult to interpret the low fat reserves and other characteristics seen in animals collected only at the end of the second winter.

I have heard occasional claims by bush pilot-guides that they have seen discrete populations of sheep almost totally disappear from their traditional range. Mr. Jack Wilson, Gulkana Airfield, has told me that there were about 300 sheep in the Hanagita Mountains of the Chugach Range in the late 1950's but that they had disappeared in the early "sixties." On August 9, 1963, on our way back in a Supercub from the Upper Chitina River, we saw only 15 sheep on a half-hour swing through the Hanagita Mountains in what had previously been well-tenanted habitat. Mr. Wilson had no idea where the herd might have moved.

B. Gastro-intestinal Helminths.

The published literature treating the gastro-intestinal helminths of *Ovis dalli* is nearly nonexistent. Philip (1937) reported finding the larval stage of *Taenia hydatigena* Pallas, 1766, in an animal taken in the Alaska Range near Isabell Pass. Becklund and Senger (1967) mistakenly included a reference to this parasite in Alaska in a checklist of parasites of Rocky Mountain bighorn sheep. It was determined later (Becklund, personal communication) that the specimens indeed had been taken from a wild sheep killed near Mt. Hayes in the Alaska Range and deposited in the U. S. National Museum Helminthological Collection. The collector obviously misidentified the host.

Rausch (1951) reported that no helminths were found in the "few animals examined" from the Anaktuvuk Pass area of the Brooks Range. The results of our observations on helminth eggs in fecal pellets collected in the Brooks Range and on the helminth burdens found at necropsy in about seventy Dall sheep elsewhere in Alaska suggest that Rausch may not have been able to perform his helminthological necropsies with enough care under field conditions.

Gibbs and Fuller (1959) reported finding the anoplocephalid tapeworm, *Wyominia tetoni* Scott, 1941, in Dall sheep in the Yukon Territory. Mr. Norman Simmonds, Canadian Wildlife Service (personal communication) informs me that they have observed helminths in Dall sheep taken in the Northwest Territories.

In our current studies on the Crescent Mountain population on the Kenai Peninsula, gastro-intestinal helminths were found in 41 of 48 (i.e., 85 percent) of the animals we examined. In addition, five other animals from the Kenai collected on Surprise Mountain after the decline of that herd were found by Mr. Lyman Nichols and Mr. Paul LeRoux of our department to be infected with several species of gastro-intestinal helminths. Most of the worms recovered from the animals collected from both herds are representatives of the nematode family Trichostrongylidae Leiper, 1912, a complex assemblage of genera and species which are particularly common parasites of both domestic and wild ruminants. At least eight genera and 21 species are known to infect bighorn sheep in North America (Becklund and Senger, 1967). Although we have not as yet identified and counted all of the thousands of specimens of trichostrongylids we found in the Kenai collection, it appears that there are about six genera and perhaps seven or eight species present. We also made counts on nematode eggs in fecal pellets from each animal using the same procedures described for lungworm larvae. The information on adult worms inhabitating the abomasum, small intestine and caecum and large intestine will be considered separately below. No helminths were found in the rumen, liver or other parts of the digestive tract not indicated above.

1. Abomasal Worms

Thirty-five of 48 animals collected on Crescent Mountain and four of five collected on Surprise Mountain were infected by stomach worms.

These included *Marshallagia marshalli* (Ransom, 1907), one or more representatives of the genus *Ostertagia* Ransom, 1907, and *Telodorsagia davtiani* Andreeva and Satubaldin, 1954. The latter species has been reported from an Alaskan reindeer (Becklund, 1962) and I have seen it in caribou taken on the Alaska Peninsula.

Judging by rough estimates of total numbers of adult stomach worms in individual infections and average numbers of nematode eggs seen in fecal pellets (lumping stomach and intestinal worm eggs together), most of the infections are light (i.e., up to 1000 adults and 50-200 eggs per gram of feces) by domestic animal standards (Skerman and Hillard, 1966; Levine, 1968).

Only one animal, an injured specimen from the Cooper Landing closed area, was judged to be more than lightly infected in terms of the usual domestic animal standards. A 3/4-year-old male taken in March, 1971, harbored more than 1000 stomach worms and its fecal pellets contained 500-1200 (800 average of six samples) helminth eggs per gram. It is worth noting that the animal also was actively infected by lungworms (3 percent lung lesions and 2600 larvae per gram of feces) and was shedding the largest number of coccidial oocysts (i.e., 9800-16,800; average 14,500 per gram of feces) that we saw in any of the animals from the Kenai collection. It also harbored about 500 trichostrongylids and a few whipworms (i.e., *Trichuris* sp.) in its intestinal tract. Its marrow fat level was down to 6.1 percent and other fat storage depots were depleted. By way of comparison, two other 3/4-year-olds collected in March, 1971, from the Crescent Mountain herd showed marrow fat levels of about 55 percent each. These had heavier lungworm burdens (i.e., 17.6 and 11 percent lesion size and 4900 and 1700 lungworm larvae per gram of feces), but lower coccidial oocyst levels (i.e., 5000 and 6000 per gram). One of these had a comparable burden of stomach and intestinal worms, but was shedding only about 60 eggs per gram of feces. The other with only one-fifth as many stomach and intestinal worms was shedding about 80 eggs per gram of feces. It appears that the relatively high numbers of helminth eggs, lungworm larvae and coccidial oocysts being shed by the injured, starving lamb from the Cooper Landing closed area show the inhibiting effects of under-nutrition on the normal immune responses of the host. It also seems clear that the poor nutritional state of the animal was principally due to the impairment of its feeding activities by its injury, a broken femur. Its relatively high parasite load probably accelerated the rate at which it was declining in condition.

2. Intestinal Worms

The helminth fauna of the lower digestive tracts of ruminants is relatively more diverse than that of the abomasum and rumen. Wild sheep are no exception. According to Becklund and Senger (1967) North American bighorn sheep are host to six genera and 15 species of nematodes and three genera and four species of cestodes all of which inhabit the intestines and caeca.

Our incomplete study of the multitude of specimens of intestinal and caecal helminths recovered from the Kenai sheep collection has

revealed several species of the trichostrongylid genera *Cooperia* Ransom, 1907, and *Nematodirus* Ransom, 1907; the sheep whipworm *Trichuris ovis* (Abildgaard, 1795) and a few cestodes of the genus *Moniezia* Blanchard, 1891. Although we have commonly found substantial numbers of the caecal pinworm, *Skrjabinema* Vereshchagin, 1925, in recent studies on sheep collected at the Dry Creek study area, in the Alaska Mountain Range, we failed to see these in any of the Kenai sheep. I am inclined to suppose that we may have overlooked an occasional, light infection of this small worm. Further comments on the various kinds of helminths we encountered are presented separately below.

a. Trichostrongylids

About 40 percent of the sheep from the Crescent Mountain study area were lightly infected with intestinal trichostrongylids averaging 60 worms per infection. The heaviest Crescent Mountain infection involved only 168 individuals by actual count. The injured lamb from the Cooper Landing closed area, which was discussed in the preceding section on stomach worms, harbored about 440 worms by actual count. In our present studies on sheep from the Dry Creek study area we have counted up to 2468 intestinal trichostrongylids in an animal also harboring lungworms and several thousand other gastrointestinal helminths. This animal, a 7-year-old, pregnant ewe, was collected in early May, 1972, and was considered to be in generally good condition for late spring.

Four of the five sheep collected in April, 1970, from Surprise Mountain after the decline were found by the sheep study leader, Mr. Lyman Nichols, and his assistants, to harbor from one to seven intestinal trichostrongylids. These necropsies probably did not reveal all of the helminths actually present.

b. Whipworms

About 45 percent of 48 Crescent Mountain sheep harbored an average of about thirteen whipworms each. The heaviest infection, seen in a yearling female in late April, 1971, involved about 150 worms. Our specimens are most likely examples of *Trichuris ovis* (Abildgaard, 1795) which is widely found in wild ruminants. They normally occur only in small numbers and are not known to be significantly pathogenic even in domestic animals (Levine, 1968).

Two specimens were recovered from one of the five animals taken from Surprise Mountain.

I have seen whipworm ova in pellets collected on various other sheep ranges in Alaska and we are commonly finding light infections in animals collected on the Dry Creek study area.

c. Tapeworms

Several species of anoplocephalid tapeworms occur in North American bighorns (Becklund and Senger, 1967). They are uncommon in the Dall

sheep we have examined to date. Only three infections came to light in the animals collected on Crescent Mountain. In each case only parts and pieces of strobilae and/or ova in fecal pellets evidenced the infection. One of the five animals taken on Surprise Mountain also harbored three small strobila. The species of tapeworm involved in these infections is most likely *Moniezia benedeni* (Moniez, 1879). We have seen this helminth in Alaskan black-tailed deer (*Odocoileus hemionus sitkensis*) where it was associated with an obvious case of diarrhea. The parasite is worldwide in distribution in wild and domestic ruminants in which it generally infects only young animals. The four infections in Dall sheep cited above all involved young animals from 6 to 24 months of age. This parasite is accused of being pathogenic in heavier infections.

Another anoplocephalid, *Wymonia tetoni* Scott, 1941, apparently also occurs in Alaskan Dall sheep. As noted earlier, this worm has been reported from Yukon Dall sheep. Specimens have been recovered from a Dall sheep taken in the Chugach Mountain Range in Alaska (personal communication, Dr. R. L. Rausch, Arctic Health Research Center). This parasite was first recorded in bighorn sheep in Wyoming and is now known to also occur in this host in Arizona, British Columbia, Montana and New Mexico (Becklund and Senger, 1967). It normally inhabits the bile ducts of the liver and the gall bladder, but the major part of the strobila may be found in the small intestine. Apparently it is not known to be significantly pathogenic.

C. Coccidia.

Although species of the coccidian genus *Eimeria* Schneider, 1875, are well known pathogens of domestic sheep and are commonly known parasites, if not pathogens, of many species of wild sheep, hardly anything is known of this class of parasites in Dall sheep. Since 1961 I have examined fecal pellets from several Alaskan sheep ranges and have found coccidial oocysts more or less commonly in each area including the Kenai, Chugach and Wrangell mountains and the Alaska Range. Only relatively recently have we had at our disposal a microscope suitable for interpreting the anatomy and identifying oocysts to species, but that task has not yet been accomplished for the large amount of material on hand.

There is only one bona fide publication of which I am aware that includes information on coccidia of Dall sheep. Uhazy et al. (1971) reported the oocysts of *Eimeria ahsata* Honess, 1942, and *E. crandallis* Honess, 1942, in a small series of fecal pellets collected on Dall sheep range in the Northwest Territories. They made no further comment concerning these infections. However, Mahrt and Sherrick (1965) have seen fatal infections of *E. ahsata* in domestic, feedlot lambs and one must suppose that infections of high enough intensity would also be pathogenic in wild lambs.

Thirty-eight of 48 Crescent Mountain sheep were found to be infected with one or more species of *Eimeria*. These infections ranged from 125-12,100 (average 2300) oocysts per gram of fecal pellets. The injured

lamb collected on Surprise Mountain showed the highest number of oocysts, 14,500 per gram of fecal pellets. This maximum number is about 10 percent of the value associated with abnormally soft pellets in two captive bighorn lambs (Uhazy et al., 1971). While some species of coccidia are extremely pathogenic in domestic lambs kept in crowded conditions, it is probably seldom that coccidia act other than as immunizing agents in wild sheep. Still, subclinical infections are extremely common and pathogenic infections no doubt can occur whenever a lamb gets a big enough dose of oocysts in its first exposure.

D. Lump Jaw.

An advanced case of so-called "lumpy jaw" is a spectacular lesion. Extensive erosion, fenestration and swelling of one or both mandibles and loss of teeth, sometimes all of the molars, are remarkable lesions to more or less commonly see in sheep which are apparently otherwise in relatively good condition.

The published literature contains more detailed information on this disease condition than on any other in Dall sheep. Murie (1944) reported that 213 of 829 Dall sheep skulls picked up in McKinley Park showed unmistakable signs of mandibular and/or maxillary disease. So-called "lumpy jaw" lesions were seen in approximately equal numbers of rams (98) and ewes (105) and also in 10 yearling animals.

The first report on this disease condition in North American wild sheep of which I am aware is that of Blair (1907) who found signs of the disease in three of six Stone sheep skulls picked up in British Columbia. Sheldon (1932) reported that all eight specimens of Stone sheep collected by him in British Columbia showed signs of jaw disease. Couey (1950) reported several, severe cases in adult Rocky Mountain bighorns in Montana.

We have been soliciting mandibles from Alaskan hunters and guides for several years, and have records on 125 sheep not including those taken in our scientific collections. While 65 of these show obvious signs of mandibular disease, we do not feel that this truly represents the normal prevalence of this disease condition. Unfortunately, in many instances only obviously diseased mandibles are brought into us.

We have data on 46 of the 48 animals collected during the Crescent Mountain study. Sixteen of the 46 showed obvious signs of either abnormal swelling or erosion of the mandibular bony tissue and/or loss of one or more teeth. Whether or not these few observations accurately represent the prevalence of "lump jaw" and related disease conditions in the Crescent Mountain sheep is hard to say.

A Case of Lump Jaw

Although we have already briefly considered some of the information on the lump jaw-pneumonia Case #690, it seems worthwhile to comment on it further. This animal was originally selected during the Dry Creek tagging operation as one of two or three to be brought into town for

helminthological studies. At the time it was collected it was noted that its right mandible was markedly swollen, which added to our interest in it.

"Six ninety" and its companion "Seven forty two," another ewe, were held in a large, shaded pen at the Arctic Health Research Center animal facility on Ft. Wainwright. The pen had been used several months previously to hold pigs and was situated near caged black bears, wolves and coyotes, and a team of sled dogs. It is hard to say whether or not the relative closeness of these predators constituted a significant stress on the sheep. They only showed obvious signs of nervousness and alarm when humans closely approached or entered their pen. During the first three weeks of July, in order to allow them to accommodate to new surroundings, we did not attempt to handle them other than during a preliminary, general examination by Dr. Maury Hamlet, Veterinarian, Aeromed Laboratory, Ft. Wainwright. They were fed Purina Dairy Chow and watered daily. However, in spite of what appeared to be congenial circumstances, "Seven forty two" sickened on August 3 and, as noted in an earlier section of this report, died on the following day from a condition diagnosed as pneumonia by Dr. Hamlet.

It was obvious from the start that "Six ninety" was bothered by its infected jaw. It repeatedly worked its mandibles back and forth, laterally, about every half minute or so, probably in response to the several loose molars which were evident postmortem. In mid-July an abscess fenestrated and began draining from the ventral, mid-molar region of the swollen right mandible. Because of the continued, extreme skittishness of "Seven forty two," who was still alive at that earlier date, and "Six ninety" we decided not to attempt to take periodic samples of blood for other studies we had in mind. However, we finally decided that we should attempt to obtain specimens from the draining abscess on "Six ninety" for bacteriological studies. Accordingly the animal was caught and manually restrained while the abscess was explored with a sterile hypodermic syringe and large gauge needle with which a quantity of yellowish pus was finally obtained. The specimen was referred to Dr. Lucille K. Georg, Center for Disease Control, U. S. Public Health Service, Atlanta, Georgia. The results of the bacteriological studies on the specimen from "Six ninety" were unexpected.

N No evidence of an *Actinomyces* sp., neither typical sulfur granules which give the purulent material formed in *Actinomyces* infections a yellowish color as we saw in our specimen nor the organism itself, was seen. As we noted earlier in the brief description of the terminal pneumonia suffered by "Six ninety," only two species of *Streptococcus*, i.e., *S. faecium* and *S. sanguis*, were isolated.

During the following months when we were primarily concerned with helminthological studies on "Six ninety," the abscess healed over. However, the obsessive "jaw grinding" continued unabated. In mid-November it was necessary to move "Six ninety" to new quarters in the live animal compound at the University of Alaska. A small pen, about 10 x 12 feet, was constructed in one corner of a larger pen in which a flock of about

a dozen domestic sheep were being held. A small, three-sided and roofed shelter was provided. The daily care remained as before and the animal prospered. It grew a thick winter coat and displayed well rounded contours.

On about December 15, "Six ninety" appeared to be some less alert than usual, and during the next several days for no apparent reason she became progressively more lethargic. She died sometime during the morning on December 22, 1971, and with the aid of Drs. Robert Dieterich and Rollo Van Pelt, Institute of Arctic Biology, University of Alaska, an autopsy was performed that afternoon. We have already commented on the massive lesions of chronic pneumonia which quite obviously pointed out the cause of death.

Because we were also keenly interested in the "lump jaw" condition of "Six ninety," we took out the mandibles without unnecessarily disturbing the soft tissues of the affected areas. The mandibles were rapidly frozen and shipped to Dr. Lucille Georg at the Center for Disease Control for determinative, bacteriological study. I have noted earlier in this report the main details of these studies. Only two organisms were unequivocally identified, i.e., *Cornebacterium pyogenes* and *Fusobacterium necrophorum*. Both of these species are common infectious agents in domestic and wild ruminants around the world, the latter being an old acquaintance, the hoof-rot agent, i.e., *Spherophorus necrophorus* with a new generic epithet. Since only one of the mandibles appeared swollen in the intact animal, we were surprised to see that both had been relatively severely affected as shown in Fig. 6.

It seems worthwhile to quote selected parts of Dr. Georg's report. "Dr. Kaplan and I have examined a number of cow heads with lumpy jaw and on first examination this head appeared quite similar...We suspected the Gram + organism to be an *Actinomyces* species, even though it was morphologically atypical, and made numerous smears for direct staining with our various *Actinomyces* conjugates. These included *Actinomyces bovis*, *A. israeli*, *A. naeslundii* and *A. viscosus*. These results were negative. However, the organisms did stain with our conjugate for *Corynebacterium pyogenes*...After many studies there is no doubt in our minds now that the Gram +, pleomorphic organism is *C. pyogenes*. In fact, the organism in the original pus smears stained with the FA conjugate for *C. pyogenes* showed the same morphology as in the gram stains...Quite frankly, I don't know what these results mean. *C. pyogenes* is, as you well know, commonly found in suppurating lesions in animals. However, I am not aware that it can produce osseus lesions similar to those caused by *Actinomyces bovis*. There is a possibility that an *Actinomyces* species had been involved in an earlier stage of the disease, and subsequently disappeared. However, I am disturbed by the fact that we saw no evidence of granules... I don't believe we have solved the problem of the etiology of lumpy jaw in wild sheep."

For many years it has been supposed, for no other obvious reason, that because "lumpy jaw" in wild sheep grossly resembles the condition of the same name commonly seen in cattle, that the causation must be the

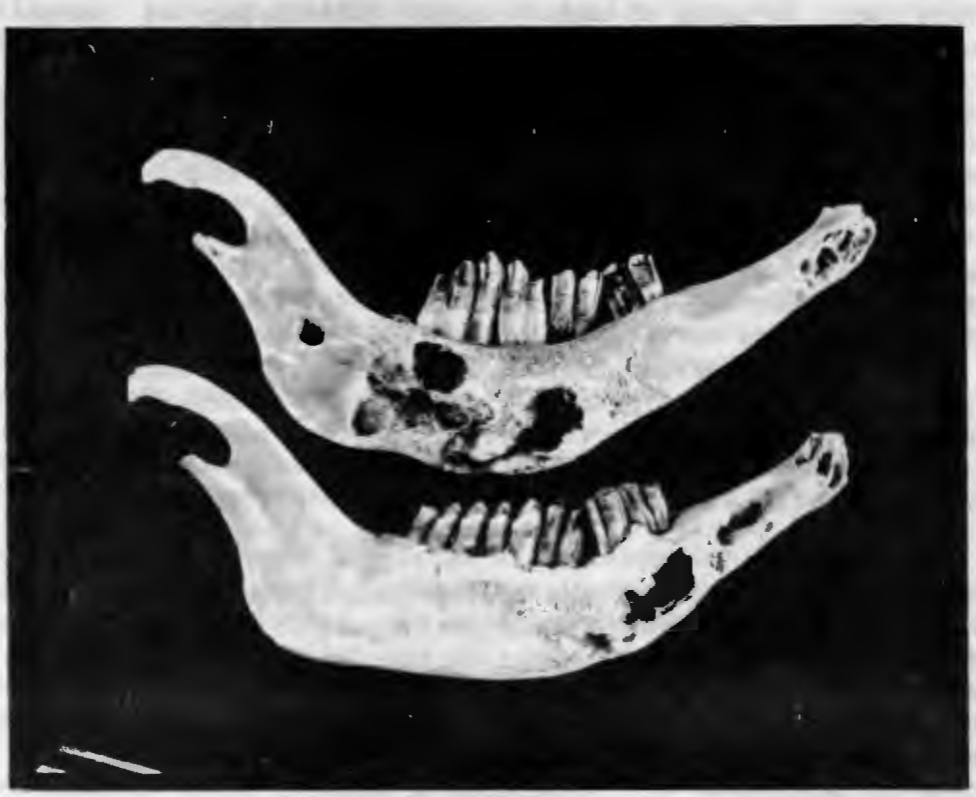


Figure 6. Mandibles from Dall sheep ewe #690 showing extensive "lump jaw" lesions.

same. At present I am unaware of any published studies in which an attempt was made to actually isolate potentially causative organisms from cases of "lumpy jaw" in wild sheep, but there well may be some. In any event, the evidence at hand suggests that there is as yet no factual basis for classifying "lumpy jaw" of Dall sheep as an actinomycosis. As the reader should have noted in the quotations from Dr. Georg's report, the absence of "granules" (i.e., yellowish sulfur granules) is the most salient, puzzling fact, in terms of understanding the etiology of "lumpy jaw" in wild ovines. If this condition in Dall sheep, etc., does indeed involve *Actinomyces* sp. sometime during the early course of the bony phase of the infection, where are the granules? We have recently sent additional material to Dr. Georg which may help resolve this question. However, it is worthwhile noting before leaving this topic that of several cases of lump jaw in Dall sheep examined by Dr. R. L. Rausch, Arctic Health Research Center (personal communication), none have shown sulfur granules. Also, Jubb and Kennedy (1963) claim that not all instances of mandibular osteomyelitis (e.g., "lumpy jaw") are caused by *Actinomyces bovis* and that a variety of nonspecific bacteria can cause osteomyelitis by spread from peridontitis. They further note that, "invasion via the gums produces typical lesions in the submucosal tissue. Extension to the periosteum causes actinomycotic periostitis and the infection may not progress further than this; *C. pyogenes* invading under the same opportunities will also produce lesions of this type in this location."

An admittedly limited review of the appropriate literature has failed to turn up much information on the significance of lump jaw to the welfare of wild ruminants. The subject is very briefly treated under the possibly erroneous heading "Actinomycosis" by Howe (1970). According to Howe, Green (1949) has claimed that lumpy jaw is a "significant mortality factor" in bighorn sheep in Banff National Park, Canada, where the disease is thought to be transmitted at mineral licks and water holes. Apparently Green (and Howe who failed to comment critically) are unaware that *Actinomyces* (if this indeed is the causative agent in lump jaw of sheep) is a common, obligatory, but not always harmful parasite which occurs in nature on the mucous membranes of the mouth and pharynx. There it occasionally becomes pathogenic and sets up disease when it obtains access to the body tissues (e.g., periosteum) and finds conditions suitable to its multiplication (Stableforth and Galloway, 1959). The likelihood is that sheep or other animals are exposed early in life when licked and nuzzled by their mothers. It seems likely that the immunologic defenses at the command of the individual are the primary factor determining whether the disease develops, and not just simple exposure. The relative rarity of lump jaw in non-ovines, e.g., moose, caribou and deer, suggests that wild sheep have an inherently increased susceptibility to the disease. Even in sheep the disease is probably not highly contagious.

It seems to me that there are two principal ways in which this chronic disease condition can significantly diminish the welfare of severely affected individuals or populations. If extensive malocclusions and/or loss of molars significantly impair mastication of forage, the

victims will not be able to efficiently utilize what they eat and will lose condition disproportionately to the prevailing seasonal, nutritional potential of the range they inhabit. The animal probably will be at a greater disadvantage on winter range where the forage is relatively coarser, less succulent and of poorer quality. It is well known in domestic cattle that lump jaw lesions "may attain a considerable size and ultimately lead to interference with mastication and progressive loss of condition" (Stableforth and Galloway, 1959).

Another way in which chronic lump jaw lesions may seriously influence the victims' welfare is that such abscesses may serve as reservoirs of infection from which other organs may be infected. In considering actinomycotic lump jaw, Jubb and Kennedy (1963) claim that "metastatic spread via the blood stream gives rise to secondary lesions particularly in such organs as lungs, liver, bone and brain." This, of course, can also occur with other bacterial agents, e.g., *Corynebacterium*, *Brucella*, etc. One cannot help but wonder whether the disseminated infection by *Corynebacterium pyogenes* seen in "Six ninety," involving not only abscesses of both mandibles but also the massive abscessation of the lungs and smaller foci of infection in the kidneys, might not be an example of such a metastasis. I have so speculated in the section dealing with verminous pneumonia.

To sum up our current knowledge of the lump jaw condition in Alaskan Dall sheep, it may be noted that it occurs in all of the sheep populations we have examined. We have seen examples of relatively severe infections in most, if not all, of these herds including the one on Crescent Mountain. The most spectacular example I have seen involved an animal taken on the Kenai by a hunter who gave the mandibles to an ex-employee of our department who took the specimen out of the state. All the molars had been lost. Additional details regarding the condition of the animal are not currently available to me. In order to more accurately assess the importance of this disease in Alaska, we must gather a good deal more information on its prevalence and intensity in our major herds over several years time. Unfortunately, we are currently able to get information on only relatively few of the thousand or so sheep killed each year.

E. Conclusions.

The Dall sheep on Crescent Mountain, and no doubt most Alaskan sheep populations, are well supplied with the various pathogenic agents (e.g., bacteria, helminths, coccidia, etc.) which are known in sheep elsewhere. If the collection of animals taken from the Crescent Mountain herd was representative of the general state of health of the herd, we may conclude that there were essentially no acute health problems during the collection period. What the chronic infections we observed may have meant in quantitative terms to the welfare of the herd is beyond the current state of the art of wildlife disease investigations. I assume that the degree of adversity was relatively slight, but it may indeed be true that sometimes, "the straw will break the camel's back." We have no reason to believe that this may have happened in this instance, but of course do not know "how close a call we may have had."

Because of our comparative state of ignorance regarding the populational effects of chronic diseases of wildlife, "parasitic" or otherwise, we cannot often assess wildlife disease problems of a chronic nature in absolute terms. Therefore, most studies are interpreted in relative terms. However, in the case of the Crescent Mountain Dall sheep population, we have no unequivocal, comparative information to which we may relate what we saw. This problem and others relating to adequacy and extensity of sampling by season, age class, sex, etc., qualify, most if not all, published studies to the point of near meaningless in terms at least of the broader considerations at hand. One cannot help but wonder, however, if such lower-level, chronic pressures may not be important, not year to year, but in long-term population trends. To my knowledge there have been few (if any?) long-term studies of the sort which might objectively measure influences more subtle than death by gunshot, climatological misadventure or fang and claw.

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