Bighorn Pneumonia Die-Offs: An Outsider's Synoptic History, Synthesis, And Suggestions

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[**Author's Note:** I'm not a veterinarian, a bacteriologist, or a molecular biologist. What I am is an inter-disciplinary-trained observer and occasional participant in our quest to solve the problem of bighorn die-offs. I am committed to inter-disciplinary or reviews in science. Without the cross-fertilization of "outside thinking" many of the breakthroughs of science, ranging from the structure of DNA to the integration of sheep behavior with harvest management, would have been longer in coming. I think an additional benefit of "outside review" is that it may encourage us to examine our present positions from differing perspectives. It may even help us to take our selves and our opinions a little less seriously and broaden our thinking in the process. I appreciate the editors of this proceeding allowing the use of an informal essay format to share these ideas. I am grateful to Dr. Karen Rudolph for details in the history of *Pasteurella* taxonomy. WH]

Abstract: The presence of pneumonia die-offs in bighorn sheep prior to European settlement of North American is unknown. With European settlement of the American West, pneumonia die-offs became the dominant factor in management and restoration of Rocky Mountain and California bighorn sheep. Early work suggested lungworm parasites were *the causal factor*, and the "lungworm-pneumonia complex" was taught as causative in wildlife and ecology curricula for decades. As bighorn sheep recovery associated with predator extirpations and prohibitions on human harvests progressed, managers realized the potential for human benefits from bighorn sheep harvests, and began to approach management of pneumonia die-offs in bighorns. Presuming parasites were causal, antihelminthic drugs were seen as the treatment. The drugs purged bighorns of parasites in laboratory conditions, but pneumonia die-offs persisted in the wild. Eventually, enough die-offs were statistically and pathologically associated with domestic sheep presence that domestic sheep replaced the "lungworm-pneumonia complex" as *the causal factor*. Managers then generally presumed that bighorn pneumonia die-offs would end if domestic sheep were excluded from bighorn ranges. Still, bighorn pneumonia die-offs were reported in bighorn populations with no documented exposure to domestics. This finding caused some tension between the "domestic-caused" (Pasteur's germ theory descendants) and "stress-caused" (Bechamp's internal environment descendants) camps of pneumonia die-off researchers. This tension has been politically exacerbated as it has involved domestic sheep grazing on public lands. Current work suggests that differing *Pasteurella*-like bacteria may account for these observations. Still, *Pasteurella* taxonomy is, at best, complicated; and recent innovative approaches to bacterial taxonomy have resulted in identifying three presumably different "*Pasteurellas*" reportedly responsible for bighorn die-offs. The highly virulent domestic sheep "*Pasteurella*" (formerly *Pasteurella haemolyticia*, is now called *Mannheimia haemolytica*) will almost certainly cause fatal pneumonia in any wild bighorn exposed to it. The other *Pasteurellas, trehalose* and *multocida* may or may not cause pneumonia die-offs depending on circumstances. Efforts to define a (or the) "hot bug" have recently turned away from traditional bacterial identification techniques toward identification based on genomic structural similarities. The appropriateness of either approach is determined by perspective. These perspectives are discussed with respect to bighorn management relevance, putting "all one's eggs in one basket" and "seeing the forest for the trees."

We don't know whether bighorn sheep populations experienced major population fluctuations before the advent of European settlers and their domestic animals. However, with European settlement of the American West, bighorn die-offs became the dominant factor in management and restoration of Rocky Mountain bighorn sheep. Wildlife biologists and wildlife-driven veterinarians have done their best to solve this problem, but the problem persists. The history of our experience with bighorn die-offs should point us to progress with this problem because we've been confidently wrong so often in the past. This history should prompt us to a cautionary and reserved commitment to our current understanding of the problem. It is not a simple one.

THE INITIAL (OBVIOUS) SOLUTION

Although the problem has become complex in concept with the passage of time, and its solution has evolved toward even greater convolution, the issue was initially perceived quite simplistically. It seemed intuitively clear to early sheep biologists that introduced domestic animals were getting the better of the remnant native bighorn populations in the competition for food. In retrospect, this is readily understandable because density-dependent forage limitation was the basic mantra of early wildlife management. For early bighorn biologists, it was basically an article of faith that "food was everything." Even today, this idea persists as the central dogma in the traditional wildlife management curriculum.

The earliest notable advocate of removing domestic livestock from bighorn ranges was the visionary biologist (or 'wing-nut' depending on one's point of view), James K. Morgan. Morgan argued stridently that, "domestic livestock had to go." More than a quarter of a century later, we can appreciate the validity of Morgan's argument as it relates specifically to domestic sheep, but for reasons, which dramatically eclipse the competition for food. In retrospect, we can also appreciate Morgan's finding that getting into barroom fights with ranchers and cowboys is not a particularly productive approach to the problem. Partly because of Morgan's confrontational approach, and partly because a more sophisticated model always seems more attractive to wildlife managers, Morgan's forage competition model was eventually supplanted by a parasitic disease explanation.

THE "LUNGWORM-PNEUMONIA COMPLEX"

When North American wildlife management emerged in the second quarter of the 20th Century, wild sheep were virtually absent from the scene. They had been long-since decimated throughout their ranges by what were then uncertain but rationally speculative factors including over hunting, competition with domestic livestock, and diseases associated with domestic livestock. As the unprecedented restoration of other North American wildlife species began with the invention of wildlife management (and its funding source), proto-bighorn biologists began to monitor bighorn population trends. They soon observed that bighorn populations appeared to be cyclic in nature with expansion phases followed by major die-offs that did not appear to be completely food-related. Veterinarians and pathologists were summoned to help.

These veterinary pathologists reported bighorn deaths in die-offs were typically due to bacterial pneumonia coincident with a huge infestations of lungworms in the affected populations. Eventually, a

Colorado parasitologist, Ron Pillmore, described the life cycle of the most common bighorn sheep lungworm, *Protostrongylis stylisi*, complete with its alternate host, a lowly snail. In the minds of the wildlife biologists of the day, the presence of an apparent abundance of lungworm parasites in sheep dying from pneumonia suggested parasites were *the causal factor*. The role of the bacteria involved in the pneumonia was simply not appreciated at this time.

[Author's Note: We now understand the pneumonias are caused by bacteria identified as "Pasteurellas," and that wild sheep apparently carry benign forms of these bacteria as a normal compliment of their pharyngeal flora. However, through the 1980s, the prevailing thinking was that ONLY domestic livestock harbored Pasteurella bacteria. The few attempts to isolate Pasteurella from bighorn sheep prior to1990 failed. Nevertheless, Alton Ward and Dave Hunter (from Idaho) persevered in the search for Pasteurella bacteria in bighorn sheep, and in about 1990 they showed bighorns normally carry benign (to them) Pasteurellas. Subsequently, some friends and I found several varieties of Pasteurella in remote Arctic populations of thinhorn, Dall, sheep (which had never been know to have a disease-related die-off); but this is getting a little ahead of our story. Up until the Pasteurella research 'bloom' in the 1990s, the presence of bacteria was considered secondary to the dramatically apparent lungworm involvement. WH]

Striking lungworm infestations were linked with die-offs, and integrated to produce an explanatory hypothesis, the "lungworm-pneumonia complex." This model held that as bighorn populations grew, parasite loads increased because of greater bighorn sheep population density, and the burgeoning lungworm populations in sheep weakened them to the point they developed an opportunistic bacterial pneumonia. This seemingly robust model was taught as an illustration of density-dependent population regulation in the wildlife and ecology curricula for decades. However, there was more to the story than lungworms and high bighorn concentrations. The bacteria involved would eventually prove to be the "wild card" in the system, but more on this later.

As limited bighorn sheep population recoveries associated with predator extirpations and the prohibition of human harvests in pristine habitats progressed, forward-thinking managers began to realize the potential for human benefits from bighorn sheep harvests. As a result, the presumably parasite-driven die-offs related to the "lungworm-pneumonia complex" in bighorn sheep began to draw the attention of sheep biologists and wildlife veterinarians. Colorado scientists, Chuck Hibler, Terry Spraker, and Bob Schmidt were pioneers in this area. Among other things they demonstrated transplacental transmission of lungworm larvae to bighorn fetuses in bighorn ewes with unusually high lungworm infections. This seemed supportive of the "lungworm/pneumonia complex" model as it seemingly explained why lamb survival was poor during and following population collapses.

Managers turn to drugs

From these data, managers inferred the obvious way to sustain higher-density bighorn populations (and increase sheep-related human benefits) was to get rid of the parasites. Subsequently, antihelminthic drugs, those that would kill the lungworms but not the sheep were seen as the way to stable sheep abundance. These drugs, in the "diazole" family, are chemical cousins of some very nasty

molecules. That's probably why they work. However, they were approved for use, and worked in domestic livestock. Soon, our Colorado friends tested these drugs on captive bighorns with lungworm infections.

The drugs clearly purged bighorns of parasites in controlled laboratory conditions, and were eventually shown to reduce transplacental transmission of lungworm larvae to fetal bighorn lambs. However, in some cases, most notably a Colorado herd where wild, free-ranging sheep were given heroic doses of antihelminthic drugs in apple pulp (and salt blocks) and lungworm infestations dramatically decreased, lamb survival did not increase. The "lungworm-pneumonia model" was not working out as well as expected.

Managers renounce drugs for bacteria

Eventually, controlled laboratory tests by our Alberta friends, Detlef Onderka and Bill Wishart, showed lungworm-free sheep would still die of pneumonia if infected with bacteria from healthy domestic sheep. The now classic "lungworm-pneumonia complex" explanation had proven, by itself, to be an inadequate explanation. Clearly other factors were involved, most probably infection with bacteria from apparently healthy domestic sheep. Nevertheless, the idea of lungworms predisposing sheep to bacterial pneumonia persists, particularly in what I shall call "lungworm/pneumonia complex" county.

About this time, Jim Bailey (then at Colorado State University, the heart of "lungworm/pneumonia complex country") steered Nike Goodson (now Stevens) toward a systematic compilation of factors related to bighorn die-offs. Nike's synthesis showed bighorn die-offs were tightly linked to the presence of domestic sheep on bighorn ranges. This finding, along with the bacterial evidence implicating bacteria from healthy domestic sheep in bighorn pneumonia, resulted in the inference that "domestic sheep bacteria" were *the causative factor*. Parasites were "out," but not forgotten. Measurement of lungworm burdens in wild sheep continues on many ranges, and interest in parasites enjoyed a recent resurgence in Dall sheep from Canada's Northwest Territories.

Compelling evidence that parasites, in the absence of domestic sheep (and their bacteria), cause major population declines in wild sheep is still lacking. Viewed in the contemporary framework of adaptive response, a parasite which kills its host is not considered very good at its job. Hence, parasitic studies seem likely to remain more an academic pursuit than one of high management relevance. Domestic sheep presence/bacteria replaced the "lungworm-pneumonia complex" as the favored factor limiting bighorn management success in the minds of many active bighorn managers, particularly those from the Northwestern U.S.

"THE GREAT BUG HUNT"

A natural intuitive reaction

Once biologists were, again convinced they knew *the cause* of the die-offs (this would be the third time-first it was parasites, then parasites predisposing bighorns to bacterial infections), the first order of business became identification of the bacterial species presumed responsible for bighorn pneumonias. It appeared normal, healthy domestic sheep carried bacteria that were deadly to the bighorns. Thus began "the great bug hunt."

At the outset, we should note that the rationale for "the great bug hunt" has never been clearly stated, nor achieved a broad consensus. It just 'kind-of happened.' The great bug hunt has been,

as was density-dependent limitation before it, more of an intuitive reaction than a rational decision. I define this as a forgivable failing; it should have been expected. After all, identifying the pathogen has been the traditional, and largely successful, approach to disease management for livestock and humans for the almost 150 years since Louis Pasteur. Naturally, it was the most obvious path to pursue.

In humans and livestock, disease control prescribes identification of disease agents so they can be cultured, a vaccine produced (we hope), and the disease managed or eliminated through immunological manipulation. While the prospect of a vaccine has always been inferred from the great bug hunt, the bighorn management community has never actually faced the issue of whether this is feasible from the vaccine development side, the vaccine administration side, or the ethical perspective. Nevertheless, we're in the great bug hunt.

Perhaps ironically, the object of the great bug hunt, *Pasteurella* is Louis Pasteur's namesake. The irony, it is that the "great bug hunt" is not only for Pasteur's taxonomic namesake, but is driven by his intellectual legacy, the germ theory of disease. Understanding this connection and our present situation requires some historical review I consider relevant. It seems to have passed from common knowledge

An old argument not yet settled

When first proffered, Pasteur's germ theory of disease was one of two major contending explanations for the disease state. Pasteur's major competitor was a fellow named Bechamp. Pasteur championed the germ theory of disease; Bechamp argued the disease state resulted from an imbalance in an organism's "interior environment." Pasteur won. Demonstration of the germ theory was straightforward and simple in the 1860s. We still struggle with the influence of stress and physiological or immunological compromise in relation to the disease state. Bechamp is all but forgotten, but his idea is slowly gaining credibility after 150 years.

Regional 'side choosing' in the old argument

Despite the empirical triumphs of Pasteur's germ theory of disease, the niggling observation that not every bighorn sheep herd exposed to a domestic sheep perishes (plus the fact that some bighorn populations experience pneumonia die-offs in the apparent absence of domestic sheep) has divided modern students of bighorn die-offs into the same basic camps championed by Pasteur and Bechamp. The divergence appears to be regional, and corresponds roughly with what I'm willing to call "lungworm/pneumonia complex country."

The "Pasteurites" (strict germ theory types) are primarily located in the Pacific Northwest while the intellectual descendants of Bechamp (those who advocate predisposing stressors) are further toward the east, primarily in Colorado and Wyoming, the region where the work on the lungworm/pneumonia complex dominated research for almost 40 years. The 'germ theory biologists' rely most heavily on the definitive killing assays done by Bill Foreyt at Washington State. Bill has repeatedly demonstrated, and published, accounts of penned bighorns dying of *Pasteurella* pneumonia following domestic sheep introduction to their pens. The 'predisposing stress' biologists don't deny these results, but are somewhat skeptical of their universal

application. The 'stress camp' points to its more inferential studies of stressors interpreted to cause compromised immunocompetence as well as the fact that some bighorn populations in their region have coexisted with domestic sheep for decades. Still, the 'germ theory' school maintains the upper hand, probably for the same reasons Pasteur prevailed almost 150 years ago. Gathering supportive data for the germ theory remains direct (either the bighorns die or they don't); 'stress research' is more difficult and inferential. Part of this disagreement could result from the differing camps working with differing pathogens.

These two schools of thought don't seem communicate productively. I'm certain the researchers read and relate to their colleagues results, but they don't seem to be greatly influenced by them. The more serious part of this separation results from the involvement of field managers and their propensity to take sides in a diversity of approach and opinion they may not fully understand. The 'germ theory' folks, having a well-defined loyalty to Pasteur's legacy, adhere to the rational management dictum that "domestic sheep must be excluded from bighorn ranges." This amounts to a philosophically simple but culturally complex philosophy of quarantine to favor bighorn sheep. In apparent contrast to the 'germ theory school,' the 'stress school' is less willing to postulate a simple quarantine will solve the problem. This school argues stressors in addition to domestic sheep presence probably won't be adequately managed by simply separating bighorns from domestic sheep.

The quarantine issue becomes complex and politically relevant because of the former economic, and remaining cultural, importance of the domestic sheep industry in the American West. The domestic sheep industry, fighting for its existence because of its inability to compete on the world domestic sheep products market, is looking for all the help it can get. Typically, the industry opposes any limitations on its ability to compete in a difficult economic situation. As a result, the industry seizes on this divergence of scientific perspective with the argument that that the scientists don't seem to be able to agree, so their interest, domestic sheep, should not be considered harmful to bighorns.

Unfortunately, the issue of whether or not to quarantine bighorns (particularly by excluding domestic sheep grazing on the public lands) leads to some resentment in the bighorn management community as well. The political power of the domestic sheep industry in the 'germ' and 'stress' regions is variable, and the differences over the necessity of eliminating domestic grazing (the radical quarantine) lead the 'germ' folks to consider the 'stress' folks less as colleagues with a differing approach, and more as 'domestic sheep sellouts.' This is not helpful.

As indicated above, the confusion is enhanced by the fact that these differing schools appear to be looking at differing bacterial species. The 'germ theory school' work focuses on the most deadly bacterium it can isolate, the present fruits of 'the great bug hunt.' This 'bad bug' has come to be known as *Mannheimia haemolytica*, a separate genus and species from the traditionally-studied '*Pasterurellas*.' Research in the 'stress school' has focused on the more traditionally classified '*Pasteurellas*,' particularly the species, *trehalosi* and *multocida*.'

> [Author's note: I realize I'm generalizing a bit here. The most highly publicized bighorn die-off in recent history, the Hells Canyon die-

off, is presently thought (by the involved biologists to have been *Pasteurella* (not *Mannheimia*)-driven. Nevertheless, the Hells Canyon program clearly focuses on the alleged non-Mannheimia bacterium within the context of Pasteur's germ theory of disease. WH]

Adventures in bacterial taxonomy

The first step in identifying bacteria is to obtain a presumably pure culture of the bacteria and look at it under a microscope to see if it rod-shaped, round, filamentous, has flagella etc. Once this is known, a cell-wall stain, called Gram's stain is applied. The bacterium responds by developing either a blue or a red color depending on the structure of the cell wall. Then the fun really begins because the microscopic examination and Gram's staining separate bacteria only into very broad categories.

The classic approach to bacterial identification is to take the presumed pure culture, and see what it eats. On the basis of the foods (metabolites) the bacterium can process, it is further sorted to genus and species. There are at least two inherent assumptions in this approach to identification. The first is that the culture is pure. Empirically, this assumption seems justified provided adequate micro technique was practiced in the separation procedures. The second assumption is that the bacterial culture is genetically and phenotypically stable with respect to the metabolites it can process.

The assumption of genetic or metabolic stability is apparently open to question. About 105 years after Pasteur, three other French microbiologists won the Nobel prize for description of the "enzyme activation" phenomenon in *E. coli*. These Frenchmen (Jacob, Lwoff, and Monod) found that some cultures of *E. coli* could process lactic acid when they encountered it in their culture media, and some could not. Classically, this would have been adequate grounds for separating the two cultures as different species. However, the Frenchmen found that after being exposed to lactic acid for a generation or two, the cultures, which formerly couldn't metabolize it, developed the same ability to use lactic acid as the other cultures. This meant that either the culture had always had the latent (genetic) ability to metabolize lactic acid, or it had somehow acquired the ability. Jacob, Lwoff, and Monod eventually concluded the latent ability was there all along, and was just expressed when the metabolite stimulated their cultures to express the gene required to produce the enzyme to use lactic acid. I've never heard this work referenced in wildlife bacteriology, but since it won the Nobel Prize, I figure it must have been good science. For me, this raises some questions about the confidence we should have in classic bacterial identification.

With respect to domestic sheep bacteria that can kill bighorn sheep via pneumonia, the classic identification originally came down to genus, *Pasteurella*, and species *haemolytica*. Unfortunately, this wasn't an adequate functional description because some *Pasteurella haemolytica* cultures could kill bighorns and some couldn't. 'Bug hunters' figured this meant our system of what foods a bacteria could eat wasn't detailed enough to allow us to sort the benign *Pasteurellas* from the killers. Alternately, we have to face the possibility that the bacteria, as we had identified them to genus and species, didn't "breed true" like species of more complex organisms. That is, they may have been practicing enzyme induction or have acquired the ability to kill bighorns using some other trick our sorting system couldn't identify.

The first presumption was that there were differing strains, including the 'hot bug,' we could identify through more

complex testing. One of these tests (called serotyping-because it is based on immune serum protein reactions) was used to identify very specific proteins on the surface of the bacteria as a means of sorting for the killer. This work was done with bacteria cultured from domestic sheep, and offered some promise. Eventually, Alton Ward a bacteriologist working in a domestic-sheep driven research facility, expanded on serotyping, melding it with more-detailed classic metabolic identification to produce an elaborate, second-order metabolic sorting system called biotyping. Al's lab was just down the street from Idaho's wildlife health lab, and a collaboration between the domestic-driven research unit and the wildlife folks began. Using his system, Al made the first attempts to identify the bacteria from bighorn die-offs. His work seemed to indicate there were, indeed, differing *Pasteurella* strains. It seemed Ward had been able to sort some "bad" strains of *Pasteurella haemolytica* from the benign ones. However, clear identification of 'the killer strain' proved yet-elusive. The bighorn-derived killers didn't sort as accurately as the strains from domestic sheep because they had differing (often more, as I understand it) specific surface proteins identified by the serotyping procedures. That is, instead of being identifiable as simply "A2," the common bighorn strain, they might type out as "A1, A2, or A7."

Here we should note that most of the work done on *Pasteurella* has been done by the domestic industry for which the bacteria represents problems. The domestic sheep issues with 'bad *Pasteurellas*' relate to decreased profits for ranchers and farmers. They are not basic life or death issues as in bighorn sheep. Consequently, the domestic industry has been involved in a decades-long quest to develop a vaccine to use against *Pasteurella* for economic reasons. This may be important to us because the agricultural researchers, who developed serotyping, re-used their *domestic* sheep cultures in an attempt to further identify their problem species. They did this by looking at the genetics of these domestic sheep bacterial cultures. Their method of sorting beyond sero- and bio- typing was to look at the DNA of differing cultures. When they did, they found some significant DNA differences between what had been called *Pasteurella haemolytica* and the other *Pasteurella* species, *trehalosi* and *multocida*. Consequently, they proposed a change in name from *Pasteurella haemolytica* to *Mannheimia haemolytica* based on basic DNA differences between the 'new' *Mannheimia*, and the 'old' *Pasteurella trehalosi*, and *Pasteurella multocida*.

> [Author's Note: I don't know if all the wildlife managers involved in this issue have been able to keep up with these complex, 'out-of-discipline' changes. On the chance they haven't, we should note that, while we broadly labeled the bighorn killer bacteria as *Pasteurella haemolytica*, and while domestic sheep strains formerly called by this rather broad name are now called *Mannheimia*; it may be questionable to presume the 'great bug hunt' has come to a definitive and successful conclusion because of the DNA-driven name change. We can concur with the domestic sheep reclassification, and call all previously bighorn-derived *Pasteurella haemolytica* cultures "*Mannheimia*," if we so choose. However, we should realize no bighorn-derived strains have been retyped using the DNA system the domestic industry researchers used in their re-designation. WH]

Keying on the successful reclassification of the old *Pasteurella*

haemolytica to *Mannheimia haemolytica*, bighorn 'bug hunters' have turned to the more modern and trendy DNA analysis. Here it is important to note that the assumption of metabolic (i.e. genetic) stability is still driving this portion of the great bug hunt.

I find this a bit unsettling because, plainly put, bacteria (like *Pasteurella/Mannheimia*) don't do sex the same way we do. That is bacterial DNA is of less certain origin than in life forms (like humans) that reproduce sexually. Relatively recent bacterial research shows some bacteria routinely transfer DNA by mechanisms called "plasmid transfer" (a kind of wholesale DNA swapping), "conjugation" (which is roughly the same way we do sexual reproduction), "transduction" (a form of viral DNA introduction), and a catchall called "transformation" (which means bacteria may incorporate any DNA they find lying around their environment). Additionally there is a phenomenon called "genetic splicing" wherein it seems as though proteins may "back code" for DNA. This is, of course, backwards to the prevailing model of gene action where DNA serves as a template for RNA, which serves as a template for protein synthesis. There's a whole 'brave new world' out there which I think should compel caution in "putting all our eggs in the DNA basket."

[Author's Note: I realize few, if any, of us were taught about this in school. It's new. If you want to catch up, two relatively understandable references are Barry Commoner's article "Unraveling the DNA Myth" in the February, 2002 *Harper's Magazine*, and a technical paper by John Maynard and Noel Smith called "The Genetic Population Structure of Pathogenic Bacteria." It's on pages 183-215 in the Oxford University Press Publication (1999) called Evolution in Health and Disease, edited by Stephen C. Stearns. WH].

SO WHAT?

I think this is important for two reasons. First, the assumption of genetic stability, when married to DNA analysis, intuitively leads us to look for genetic markers to identify the "bad bugs." This means we are likely to look for (presumably stable) genetic markers in what may, in all likelihood, be an unstable bacterial genome. It seems certain the genes basic to life and function (called 'houskeeping genes') must be present in living cells, and are likely to be quite similar, if not identical for all living things. This means that if we want to 'catch the bad bug,' we'll have to find where it does its nefarious work at the genetic level. It also seems certain the bacterial strains that cause fatal pneumonia in bighorns will have DNA that serves that purpose. Obviously, these cultures will prove to have differing DNA than those similar bacteria that don't kill bighorns…IF we can find the 'bighorn killing gene." Unless, of course, the enzymes required for toxin production are products of the enzyme induction phenomenon. So, I wonder, what will be the breakthrough of demonstrating the obvious in DNA banding patterns?

Second, I suggest the DNA business, though cutting edge and quite sexy, is actually retrograde in terms of progressive taxonomy. Classic taxonomy was based on physical morphology (structure). That is, we designated species on the basis of what they "were." This system was fine for making orderly lists of plants and animals, but did not serve us well when we needed to sort at a finer level of resolution. I presume we have defined our interest in a more meticulous sorting mechanism because we must presume evolutionary selection acts at a more subtle level than

gross anatomy. The emerging approach to solving to identifying differences, which are not detectable through gross physical structure, has been use of DNA analysis. This is still a basically structural approach, which does not take phenotypic adaptation to environment as seriously as I would prefer.

Paradoxically (to me) the particular fascination of the DNA-level species sorters has focused on DNA that has no known function. This approach is considered quite useful for calculating what is known as "genetic distance" (which allows us to guess how closely differing critters may be related). However, the selective significance of variations in structural DNA is unknown. This interfaces with our interest in bacterial taxonomy because the relationship between structural DNA (and analogous, necessary to basic survival 'housekeeping genes,') and functional, i.e. for our argument, 'bighorn killing DNA' has not been defined. If we would identify the 'bad bugs' on a genetic level, doesn't certainty demand that we identify the gene that produces the toxin that kills bighorn sheep. If we could sort on the basis of that gene, AND IF bacterial DNA were as stable as mammalian DNA, we might be getting somewhere in genetically identifying the 'bad bug.' I'm uncertain of the rationality of 'the great bug hunt.'

SUMMARY

As stated above, I think it unfortunate the wild sheep community has never clearly faced the rationale for finding the "bad bug." Also, I'm uncertain there is an identifiable, genetically stable 'bad bug' because of the broad spectrum of DNA swapping possibilities among bacteria. Still, if there is a management relevant rationale (please recall our original goal was to make more, stable wild sheep populations), it must be production of a bighorn vaccine against *Pasteurella* pneumonia. The wildlife community has done what it could in our intuition or emotionally-driven effort to produce such a vaccine, but our efforts pale to insignificance when compared with those of the domestic sheep industry. It has failed to solve its "*Pasteurella* problem" despite the work of many great scientists over many decades and with the expenditure of many millions of dollars. I fear production of a bighorn *Pasteurella* vaccine is a highly unlikely event.

Furthermore, if there were a vaccine, managers would have to decide whether a "vaccine protected" sheep is desirable. For a vaccine to work, it would have to be universally administered. Would "universally vaccinated bighorns" be wild? Is having "wild sheep" important? If so, how important? These questions must be faced. If our only consideration is providing sheep for harvest or transplant, and if we could protect these sheep via vaccination, the questions would be fewer. However, if management of wild sheep in wild environments is our goal, the questions about "how much management is desirable" must be faced.

Finally, the 'germ theory' vs. 'stress theory' issue has not been solved. It has persisted at least since Pasteur and Beauchamp, and while the 'germ' folks have always had the upper hand, it appears the 'stress' camp may be gaining. Given "Murphy's Law" (that whatever can go wrong will), one would logically predict that the entire bighorn die-off syndrome is probably more complex than the presence of a simple "bad bug." The evidence supports Murphy's Law. For me, investing in the idea of a single, stable "bad bug" that could be managed through vaccination is inconsistent with what we now think we know of bacterial

reproduction. Also, I must confess to having greater confidence in Murphy's Law than in the present approaches to bacterial taxonomy and management.

MANAGEMENT IMPLICATIONS

What we do know is that bighorn sheep will certainly do better if they aren't exposed to diseases that are "new" to them. It seems practically certain that separating bighorn sheep from domestic sheep would go a long way to limiting the pneumonia outbreaks that currently dominate bighorn management. Hence, my recommendation for wildlife biologists would be to leave the bacterial adventures and vaccine development to specialists in those fields, and to concentrate on doing the best we can to humanely separate bighorns from domestics. It's not sexy, and it's not new; but it will probably do more for bighorns than the excursions into DNA, diseases, and parasites that have occupied us for the last 50 years. Wild sheep habitats must, as a first step, be secure from the introduction of modern, exotic diseases and parasites. When we have achieved this, we may rationally move on to other management concerns. The 'stress camp' probably has much to tell us, and we should probably take it seriously. For now, I think the best we can do is to secure bighorn habitats from encroachment by domestic sheep and to keep bighorn populations at relatively low densities. These are, after all, the most basic of management requirements. They may be difficult, but they aren't new.