Modeling Management Strategies for the Control of Bighorn Sheep Respiratory Disease

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ABSTRACT Infectious pneumonia has plagued bighorn sheep populations and stymied recovery efforts across the western United States for decades. Management efforts aimed at controlling the disease have met limited success. Here we present a simple, non-spatial, stochastic, discrete-time model that captures basic bighorn sheep demographics and in which we simulate the dynamics of Mycoplasma ovipneumoniae, the suspected primary causative agent in bighorn sheep respiratory disease, based on our current working knowledge of pathogen transmission and impacts. We then use the model to explore the impacts of management approaches, including augmentation, depopulation followed by reintroduction, density reduction, and test and cull, aimed at reducing or eliminating the pathogen, its transmission, or associated infection costs. Preliminary results, pending a full sensitivity analysis, suggest that test and cull (testing 95% of a herd for 1 or 2 consecutive years and removing any individuals that test PCR positive) and depopulation and reintroduction (assuming ability to only depopulate 95% of the herd) offer the best probability of eliminating the pathogen, although neither are expected to be 100% successful. Augmentation (adding 30 adult ewes), whether we assume the ewes are susceptible or immune to M. ovipneumoniae, does not increase the probability of pathogen extinction or herd recovery, and in some cases may prolong pathogen persistence and diminish herd recovery. Density reduction (randomly removing 25-50% of the herd) modestly increases the probability of stochastic pathogen extinction and herd recovery, but only if ≥50% of the herd is removed. Stochastic pathogen extinction and herd recovery is predicted to occur on occasion without any management intervention. Ultimately, decisions to manage respiratory disease in wild sheep must weigh the predicted success of the management tool against financial, logistical, ethical, and value-based considerations. Here, we aim to supply mechanistic-based predictions of the relative efficacy of currently employed or proposed tools, as well as characterize the sensitivity of these predictions to our assumptions about how the disease process works.

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