Impact of *Mycoplasma ovipneumoniae* on juvenile bighorn sheep (*Ovis canadensis*) survival in the northern Basin and Range ecosystem

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Determining the demographic impacts of wildlife disease is complex because extrinsic and intrinsic drivers of survival, reproduction, body condition, and other factors that may interact with disease vary widely. *Mycoplasma ovipneumoniae* infection has been linked to persistent mortality in juvenile bighorn sheep (*Ovis canadensis*), although mortality appears to vary widely across subspecies, populations, and outbreaks. Hypotheses for that variation range from interactions with nutrition, population density, genetic variation in the pathogen, genetic variation in the host, and other factors. We investigated factors related to survival of juvenile bighorn sheep in reestablished populations in the northern Basin and Range ecosystem, managed as the formerly-recognized California subspecies (hereafter, "California lineage"). We investigated whether survival probability of 4-month juveniles would vary by (1) presence of *M. ovipneumoniae*-infected or exposed individuals in populations, (2) population genetic diversity, and (3) an index of forage suitability. We monitored 121 juveniles across a 3-year period in 13 populations in southeastern Oregon and northern Nevada. We observed each juvenile and GPS-collared mother semi-month and established 4-month capture histories for the juvenile to estimate survival. All collared adult females were PCR-tested at least once for *M. ovipneumoniae* infection. The presence of *M. ovipneumoniae*-infected juveniles was determined by observing juvenile behavior and PCR-testing dead juveniles. We used a known-fate model with different time effects to determine if the probability of survival to 4 months varied temporally or was influenced by disease or other factors. We detected dead juveniles infected with *M. ovipneumoniae* in only two populations. Derived juvenile survival probability at four months in populations where infected juveniles were not detected was more than 20 times higher. Detection of infected adults or adults with antibody levels suggesting prior exposure was less predictive

of juvenile survival. Survival varied temporally but was not strongly influenced by population genetic diversity or nutrition, although genetic diversity within most study area populations was very low. We conclude that the presence of *M. ovipneumoniae* can cause extremely low juvenile survival probability in translocated bighorn populations of the California lineage, but found little influence that genetic diversity or nutrition affect juvenile survival. Yet, after the PCR+ adult female in one population died, subsequent observations found 11 of 14 (~79%) collared adult females had surviving juveniles at 4-months, suggesting that targeted removals of infected adults should be evaluated as a management strategy.

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2 **in the northern Basin and Range ecosystem**

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17 **Abstract**

18 Determining the demographic impacts of wildlife disease is complex because extrinsic and
19 intrinsic drivers of survival, reproduction, body condition, and other factors that may interact
20 with disease vary widely. *Mycoplasma ovipneumoniae* infection has been linked to persistent
21 mortality in juvenile bighorn sheep (*Ovis canadensis*), although mortality appears to vary widely
22 across subspecies, populations, and outbreaks. Hypotheses for that variation range from
23 interactions with nutrition, population density, genetic variation in the pathogen, genetic
24 variation in the host, and other factors. We investigated factors related to survival of juvenile
25 bighorn sheep in reestablished populations in the northern Basin and Range ecosystem, managed
26 as the formerly-recognized California subspecies (hereafter, "California lineage"). We
27 investigated whether survival probability of 4-month juveniles would vary by (1) presence of *M.*
28 *ovipneumoniae*-infected or exposed individuals in populations, (2) population genetic diversity,
29 and (3) an index of forage suitability. We monitored 121 juveniles across a 3-year period in 13
30 populations in southeastern Oregon and northern Nevada. We observed each juvenile and GPS-
31 collared mother semi-month and established 4-month capture histories for the juvenile to
32 estimate survival. All collared adult females were PCR-tested at least once for *M. ovipneumoniae*
33 infection. The presence of *M. ovipneumoniae*-infected juveniles was determined by observing
34 juvenile behavior and PCR-testing dead juveniles. We used a known-fate model with different
35 time effects to determine if the probability of survival to 4 months varied temporally or was
36 influenced by disease or other factors. We detected dead juveniles infected with *M.*
37 *ovipneumoniae* in only two populations. Derived juvenile survival probability at four months in
38 populations where infected juveniles were not detected was more than 20 times higher. Detection
39 of infected adults or adults with antibody levels suggesting prior exposure was less predictive of
40 juvenile survival. Survival varied temporally but was not strongly influenced by population

41   genetic diversity or nutrition, although genetic diversity within most study area populations was
42   very low. We conclude that the presence of *M. ovipneumoniae* can cause extremely low juvenile
43   survival probability in translocated bighorn populations of the California lineage, but found little
44   influence that genetic diversity or nutrition affect juvenile survival. Yet, after the PCR+ adult
45   female in one population died, subsequent observations found 11 of 14 (~79%) collared adult
46   females had surviving juveniles at 4-months, suggesting that targeted removals of infected adults
47   should be evaluated as a management strategy.
48
49   **Introduction**
50   The study of population dynamics is essential for managing species (Williams, Nichols &
51   Conroy, 2002). Recruitment is a crucial process for population dynamics, whereby populations
52   gain individuals through births and immigration (Pradel, 1996). Recruitment varies strongly
53   across species depending on whether they are k-selected, i.e., having fewer young with greater
54   parental investment, or r-selected, i.e., having more young with reduced parental investment
55   (MacArthur and Wilson 1967). For k-selected species such as large terrestrial herbivores, annual
56   adult survival tends to be relatively high with little variation. In contrast, juvenile survival tends
57   to be more variable, and therefore population growth tends to be more sensitive to juvenile
58   survival parameters (Gaillard et al., 2000). Consequently, it is essential to consider variables
59   affecting the survival of juveniles when managing such species.
60   Bighorn sheep (*Ovis canadensis*) have a relatively low reproductive output
61   (Festa-Bianchet et al., 2019). Females rarely have more than one offspring per year and may not
62   achieve full reproductive potential until their 4th year (Rubin, Boyce & Bleich, 2000). Thus,
63   juvenile survival can have a significant impact on population trajectories (Manlove et al., 2019).
64   Juvenile survival is influenced by disease (Cassirer & Sinclair, 2007; Smith et al., 2014),
65   maternal body condition (Festa-Bianchet, 1998), forage quality (Feder et al., 2008), weather
66   (Douglas, 2001), genetic diversity (Hogg et al., 2006) and predation (Rominger, 2018). Disease,
67   particularly pneumonia, can have a dramatic effect on juvenile survival (Manlove et al., 2016;
68   Garwood et al., 2020). More broadly, respiratory disease likely caused the decline of bighorn
69   sheep across western North America and continues to inhibit the recovery of the species
70   (Cassirer et al., 2018). Therefore, evaluating the interaction of disease and other factors acting
71   simultaneously on juvenile survival is critical for the conservation and management of bighorn
72   sheep.
73   *Mycoplasma ovipneumoniae*, a bacterial pathogen, is considered the primary causative
74   agent of respiratory pneumonia in bighorn sheep (Besser et al., 2008; Cassirer et al., 2018).
75   Transmission of *M. ovipneumoniae* from domestic sheep (*O. aries*) and goats (*Capra hircus*) to
76   bighorn sheep is typically followed by high mortality of individuals in all age classes in non-
77   immune populations. Some survivors will clear the disease, while others remain chronic carriers
78   that continue to shed the pathogen despite often appearing relatively healthy (Besser et al.,
79   2013). Chronic carriers thus can transmit *M. ovipneumoniae* and sustain its presence within a
80   population, especially by transmission to previously unexposed juveniles (Plowright et al.,

81   2013). This infection pattern in bighorn sheep has been observed in multiple regions, including
82   northeastern Oregon, Idaho, Washington, Nevada, and South Dakota (Cassirer et al., 2018;
83   Garwood et al., 2020). Immunity to *M. ovipneumoniae* is thought to be strain-specific, with
84   novel strains resulting in new waves of infection (Cassirer et al., 2017).
85   Population-level responses to *M. ovipneumoniae* outbreaks, however, may vary widely.
86   Considerable variation has been observed in levels of all-age mortality at first contact,
87   subsequent adult survival, and juvenile survival in following years among populations and across
88   evolutionary lineages and habitats (Cassirer et al., 2018; Dekelaita et al., 2020). That variation
89   has been hypothesized to stem from numerous causes, including strain virulence (Kamath et al.,
90   2019), nutritional factors such as forage quality and population density (Dekelaita et al., 2020),
91   stochastic factors such as the presence of chronic carriers (Cassirer et al., 2018; Garwood et al.,
92   2020), genetic diversity of host populations (Cassirer et al., 2018), and phenological differences
93   resulting in different patterns of aggregation, contact, and dispersal (Cassirer et al., 2018).
94   Indeed, bighorn sheep inhabit ecosystems ranging from the arid deserts of northern Mexico and
95   the southwestern United States of America to the frigid northern Rocky Mountains of Alberta
96   and exhibit significant phenotypic variation and evidence of local adaptation (Wehausen &
97   Ramey II, 2000; Wiedmann & Sargeant, 2014; Malaney et al., 2015). Currently, three subspecies
98   of bighorn sheep are recognized, the desert bighorn sheep (*O. c. nelsoni*), the Rocky Mountain
99   bighorn sheep (*O. c. canadensis*), and the Sierra Nevada bighorn sheep (*O. c. sierrae*)
100 (Wehausen & Ramey II, 2000; Wehausen, Bleich & Ramey II, 2005). Previously, other
101 subspecies were recognized (e.g., Peninsular bighorn sheep, *O. c. cremnobates,* and California
102 bighorn sheep, *O. c. californiana*) (Cowan, 1940). Debate remains about whether those putative
103 lineages reflect important independent evolutionary trajectories and important local adaptation
104 (Buchalski et al., 2016; Bleich, Sargeant & Wiedmann, 2018; Barbosa et al. in review).
105   Previous studies of population dynamics in the presence of *M. ovipneumoniae* suggest
106 that disease dynamics should be evaluated across lineages and ecosystems. For instance, Rocky
107 Mountain bighorn sheep in the Hells Canyon system of Idaho, Oregon, and Washington occupy
108 relatively continuous habitat with cold winters (Cassirer & Sinclair, 2007). In this system, *M.*
109 *ovipneumoniae* tends to persist for long periods, resulting in constant disease in juveniles
110 (Cassirer et al., 2018). The same pattern of infection was also observed in Rocky Mountain
111 bighorn sheep in the Black Hills of western South Dakota and eastern Wyoming (Smith et al.,
112 2014; Garwood et al., 2020). In the Mojave Desert of the southwestern United States of America,
113 desert bighorn sheep occupy pockets of isolated mountain ranges surrounded by low-lying desert
114 but linked by inter-mountain movements, resulting in natural metapopulations (Bleich et al.,
115 1996). *M. ovipneumoniae* in this system appears to have been present periodically (Shirkey et al.
116 in press), and impacts on adult survival and juvenile recruitment appear to be highly variable
117 (Dekelaita et al., 2020).
118   Bighorn sheep in the northern Basin and Range ecosystem (including parts of
119 southeastern Oregon, southwestern Idaho, and northern Nevada) occupy transitional habitats
120 between the Mojave and Rocky Mountain systems and have a unique history. Native bighorn

121 sheep from this region was considered the "California" subspecies (*O. c. californiana*; Cowan,
122 1940), spanned from the Sierra Nevada of California north to British Columbia. In Oregon, all
123 native bighorn sheep were extirpated by 1945 (Oregon Department of Fish and Wildlife, 2003).
124 Subsequently, morphometric and DNA analysis of the extinct native Oregon populations resulted
125 in those populations being reassigned to the Great Basin desert form of the desert bighorn sheep
126 (*O. c. nelson*; Wehausen & Ramey II, 2000). Bighorn sheep now existing in Oregon all stem
127 from translocations and are managed as two lineages: Rocky Mountain bighorn sheep were
128 introduced to northeastern Oregon, and "California" bighorn sheep were introduced to potential
129 or former bighorn sheep habitat in other areas of the state using individuals from British
130 Columbia. Both lineages are formally considered Rocky Mountain bighorn sheep subspecies at
131 this time (*O. c. canadensis*; Wehausen & Ramey II, 2000). Most of the restored populations in
132 Oregon were sourced from a single translocation of 22 bighorn sheep from British Columbia in
133 1953 (Olson, Whittaker & Rhodes, 2013). However, in 2000 and 2001, as part of an
134 experimental effort to increase population genetic diversity and improve demographic
135 performance, two populations in southeastern Oregon received augmentations of translocated
136 bighorn sheep ultimately derived from different source populations in British Columbia (Olson,
137 Whittaker & Rhodes, 2012). "California" bighorn sheep were also introduced to northern Nevada
138 in 1972 from multiple source populations (NDOW, 2001; Olson, Whittaker & Rhodes, 2013),
139 and dispersal from these populations into southeastern Oregon has been observed (ODFW,
140 unpublished data). "California" bighorn populations in southwestern Idaho and northern Nevada
141 share similar histories, although translocations to northern Nevada relied on a larger number of
142 source populations than Oregon.
143   Bighorn sheep habitat in the northern Basin and Range ecosystem exhibits a
144 metapopulation-like structure, where bighorn uses discrete patches of steep escape terrain
145 separated by vast areas of grassland or sagebrush. Due to their demographic history and spatial
146 distribution, bighorn sheep in southeastern Oregon and northern Nevada have low genetic
147 diversity (Olson, Whittaker & Rhodes, 2013; Malaney et al., 2015) compared to levels measured
148 in studies of Rocky Mountain bighorn sheep in the Rocky Mountains of Colorado (Driscoll et al.,
149 2015) and desert bighorn sheep in the Mojave Desert of California (Epps, Crowhurst &
150 Nickerson, 2018). Bighorn sheep in the northern Basin and Range ecosystem also experience
151 different phenology of forage plants than observed in the desert or Rocky Mountain systems.
152 Thus, disease dynamics may be expected to differ, as well.
153   Although cause-specific mortality in bighorn sheep juveniles has been widely studied in
154 other systems (Smith et al., 2014; Cassirer et al., 2018; Cain et al., 2019; Garwood et al., 2020),
155 it has not been evaluated in "California"-managed bighorn sheep. In this study, we evaluate the
156 distribution and influence of *M. ovipneumoniae* on 13 "California"-managed bighorn sheep
157 populations in southeastern Oregon and northern Nevada, and investigate juvenile mortality in
158 relation to forage qualify and genetic diversity. Although presence of *M. ovipneumoniae* had
159 previously been verified in at least one population, the effect of respiratory disease on the system
160 was unknown. Genetic diversity of populations in this system likewise was unknown, although

161 anticipated to be low, and forage quality was expected to vary widely given the broad range of
162 elevations used by bighorn sheep and the strong influence of precipitation in this semi-arid
163 system. We used telemetry and field-based observations to monitor the juveniles of GPS-collared
164 adult females to estimate semi-monthly survival probability over a 3-year period. We
165 hypothesized that survival of juveniles would be influenced by disease, nutrition, and genetic
166 diversity. We predicted that the probability of 4-month juvenile survival would be lower in
167 populations that (1) were exposed to *M. ovipneumoniae*, (2) had lower expected heterozygosity,
168 and (3) that experienced lower forage quality, as indicated by pre- and post-parturition
169 normalized differential vegetation index (NDVI). Additionally, after observing the mortality of
170 an adult female suspected to be a chronic carrier in one of the study populations at the end of the
171 3-year period, we conducted a limited follow-up investigation of juvenile survival in that
172 population in the following year.
173
Materials & Methods
174
175
176 *Study area*
177   The populations of bighorn sheep we studied were located in southeastern Oregon and
178 northern Nevada, between 41.3 and 42.8 ºN, and 117.0 and 118.2 ºW (Figure 1). The entire study
179 area fell within the North Basin and Range (Level III classification of ecoregions in Omernik &
180 Griffith, 2014). Five populations in our study fell within or largely within the Dissected High
181 Lava Plateau (Blue Mountain [BSP], Bowden Hills [BHP], Rattlesnake [RSP], Three Forks
182 [TFK], Upper Owyhee [UOP], Figure 1), although BSP and RSP partly occurred within the High
183 Lava Plains ecoregion (Omernik & Griffith, 2014). The Ten Mile (TMP) population fell within
184 the High Lava Plains, while High Lava Plains and Semiarid Uplands (Omernik & Griffith, 2014)
185 dominate bighorn sheep habitat within the Trout Creek (Trout Creek – east [TCE], - south [TCS]
186 and – west [TCW], Figure 1) and Santa Rosa metapopulation (Calicos [CAL], Eight Mile
187 [EML], Martin Creek [MCK], and Sawtooth [SAW], Figure 1).
188   The Dissected High Lava Plateau and High Lava Plains ecoregions are both characterized
189 by elevated plateaus, but the Dissected High Lava Plateau contains sheer-walled canyons as well
190 as intermittent lakes, while the High Lava Plains contains isolated volcanic cones and buttes as
191 well as intermittent lakes and ephemeral streams (Omernik & Griffith, 2014). Mountains of low
192 to mid elevation, typically with steep slopes, and some ephemeral and perennial streams
193 characterize the Semiarid Uplands (Omernik & Griffith, 2014). The most common geology types
194 across all three ecotypes are basalt and rhyolite, interspersed with other rock types. The soils
195 derived from these rock types are fairly shallow and poor (Omernik & Griffith, 2014). Mean
196 precipitation across the study area is typically 22.5-35.0 cm per year, although some areas of the
197 Trout Creeks and Santa Rosa Mountains, receive significantly more precipitation (Omernik &
198 Griffith, 2014).
199   All three ecotypes contain sagebrush steppe. Big sagebrush (*Artemisia tridentata*) and
200 low sagebrush (*A. arbuscular*) are the most common woody herbaceous species, while common

201 indigenous herbaceous species being primarily made up of palatable perennial bunchgrasses such
202 as Idaho fescue (*Festuca idahoensis*), bluebunch wheatgrass (*Psudoroegneria spicata*),
203 bottlebrush squirreltail (*Elymus elymoides*), Thurber needlegrass (*Achnatherum thurberianum*)
204 and the less palatable Sandberg bluegrass (*Poa secunda*). Western juniper (*Juniperus*
205 *occidentalis*) is the most common woody plant across 3 ecotypes, typically found in rocky areas,
206 while the Semiarid Uplands are distinguished by willows (*Salix* spp.) in riparian areas, and
207 quaking aspen (*Populus tremuloides*) and mountain mahogany (*Cercocarpus* spp.) in snow
208 pockets (Omernik & Griffith, 2014). Ungulate species occurring in the study area include elk
209 (*Cervus canadensis*), mule deer (*Odocoileus hemionus*), and pronghorn (*Antilocapra americana*)
210 (Omernik & Griffith, 2014). Potential bighorn sheep predavtors in the study area include cougar
211 (*Puma concolor*), coyotes (*Canis latrans*), bobcat (*Lynx rufus*), and golden eagles (*Aquila*
212 *chrysaetos*; Omernik & Griffith, 2014).
213   The most common land-use practices are cattle ranching and cultivation of grains and hay
214 (Omernik & Griffith, 2014). Heavy grazing of these lands and suppression of natural fires has
215 led to the spread of large, uncontrollable fires and encroachment by invasive annual plants, such
216 as the cheatgrass (*Bromus tectorum*) and medusahead (*Taeniatherium caput-medusae*; Omernik
217 & Griffith, 2014). These grasses outcompete indigenous vegetation post-fire leading to the
218 domination of these grasses. Wildlife and cattle here rely on springs, wetlands, and artificial
219 water sources (Omernik & Griffith, 2014).
220
221 *History of bighorn sheep populations within the study area*
222   Reestablishment of bighorn sheep in the study area started in 1978 with the translocation
223 of bighorn sheep into the Eight Mile area of the Santa Rosas (Figure 1). The three Trout Creek
224 populations, TCE, TCS, and TCW, and RSP were established with single translocations each
225 from Hart Mountain (Table S1) in 1987 and 1992. Hart Mountain's bighorn sheep population
226 was established in 1954 with a translocation of bighorn sheep from Williams Lake, British
227 Columbia. Although bighorn sheep in TMP and UOP were translocated from various other
228 populations in Oregon, all those populations were ultimately derived from the population
229 established at Hart Mountain in 1954. Bighorn sheep in CAL, although translocated from the
230 Pine Forest Range in Nevada, are also derived from Hart Mountain. The remaining bighorn
231 sheep populations in the study were established from either a different original sources, e.g.,
232 MCK and SAW, which had stock from Kamloops, British Columbia, and Penticton, British
233 Columbia, respectively, or more than one source, e.g., EML. Two populations were established
234 by dispersal into unoccupied habitat: BSP was established with suspected dispersal events from
235 the Trout Creek populations in the mid to late 1990s (pers. comm. S. Torland, ODFW), and BHP
236 is thought to have been established by dispersing bighorn sheep from RSP. Other movements
237 observed include the dispersal of collared adult males between the Santa Rosa Mountain Range
238 and neighboring mountain ranges in southeastern Oregon, observed in 2009 and 2010 (NDOW,
239 unpublished data).
240

241 *Capture and sampling*
242   All capture, handling, and disease testing were conducted by Oregon Department of
243 Fisheries and Wildlife (ODFW) and Nevada Department of Wildlife (NDOW). Capture
244 methodology followed the recommendations of Foster (2004) and the American Society of
245 Mammalogists (Sikes & the Animal Care and Use Committee of the American Society of
246 Mammalogists, 2016). ODFW and NDOW captured, collared, and sampled adult female bighorn
247 sheep across 13 populations in southeastern Oregon and northern Nevada between January 2016
248 and February 2018 (Figure 1). Captures were conducted using a net gun fired from a helicopter,
249 with individual bighorn sheep blindfolded and hobbled once captured (Krausman, Hervert &
250 Ordway, 1985). Bighorn were brought to a centralized area at the base of their range to be fitted
251 with a telemetry collar and to collect biological samples, except where capture location was too
252 far from basecamp to transport them quickly, in which case they were field processed, at the
253 capture location.
254   Each adult female was fitted with a Vertex Survey Globalstar collar (Vectronic
255 Aerospace, Berlin, Germany). These collars provide a GPS location every 13 hours as well as
256 VHF signal and were set to report a mortality if stationary for 12 hours. Each collar had its own
257 unique VHF frequency, with the occasional duplicate placed on individuals in different
258 populations between which dispersal was deemed unlikely. The collars were also fitted with
259 colored tags with unique numbers, allowing for identification of individuals observed in the
260 field.
261   The age of each adult female was estimated from horn growth rings (Geist, 1966; Hoefs
262 & Konig, 1984). Blood was obtained via jugular venipuncture to determine pregnancy status of
263 adult females, obtain DNA, and to screen for disease. We determined pregnancy status of adult
264 females using a serum pregnancy-specific protein B (PSPB) assay (Drew et al., 2001). Pregnancy
265 testing of adult females was only conducted in the year in which they were captured; samples
266 were sent to Sage Laboratories (Emmett, ID) conducted testing.
267
268 *Diagnostics*
269   Presence of *M. ovipneumoniae* was detected with polymerase chain reaction (PCR) tests
270 using nasal, bronchial, and tympanic bullae swabs from each captured female bighorn sheep
271 (Manlove et al., 2019). Previous exposure to *M. ovipneumoniae* was determined using a
272 competitive enzyme-linked immunosorbent assay (cELISA) to detect antibodies in serum
273 (Ziegler et al., 2014). All tests for *M. ovipneumoniae* were performed at Washington Animal
274 Disease Diagnostic Laboratory (WADDL).
275
276 *Monitoring of juvenile bighorn sheep*
277   From 2016 to 2018, we conducted semi-monthly observations of all collared adult
278 females between April 1 and August 31. Juvenile identification was determined via observation
279 of physical contact between adult females and juveniles, such as nursing or bedding down
280 together. Juveniles are weaned at approximately 4-months of age (Festa-Bianchet, 1988); thus,

281 our observation period was intended to cover birth through weaning. We located adult females
282 for observation using an R-1000 telemetry receiver fitted with an RA-23K VHF directional
283 antenna (Telonics, Inc., Mesa, AZ). We conducted observations with Kowa TSN-601 spotting
284 scopes fitted with a 20–60x magnification mounted on tripods. Once an adult female was
285 confirmed to not or no longer have a juvenile, i.e., two consecutive observations where the adult
286 female was observed without a lamb, we stopped tracking that individual adult female (e.g.,
287 Cassirer & Sinclair, 2007).
288   We opportunistically located dead juveniles and collected samples, including either the
289 entire corpse, the pluck (heart, liver, and lungs), the head, nasal and ear swabs, and/or tissue
290 samples depending on the state of decomposition. Swabs were inserted into vials dry or
291 containing tryptic soy broth, and other samples put in a cooler until returning to our field base.
292 We then stored both the samples and swabs at -20 ºC until laboratory submission. WADDL
293 conducted gross- and histo-pathology on lung tissue samples and PCR tests on swabs.
294 Additionally, *M. ovipneumoniae* strain-typing using multi-locus sequence typing (MLST) tests
295 with four locus sequences, 16-23S intergenic spacer regions, the small ribosomal subunit, the
296 genes encoding RNA polymerase B, and gyrase B, was conducted on lung tissue and swab
297 samples from two of the juvenile mortalities recovered (Cassirer et al., 2017). We then compared
298 these strain-types to strain-typed samples from the Santa Rosa metapopulation (n = 6) and from
299 the Rattlesnakes (n = 2). WADDL conducted the *M. ovipneumoniae* strain-typing.
300
301 *Genetic sampling*
302   We used a combination of both blood samples (n = 125) and feces (n = 66) as sources for
303 DNA samples. Whole blood (3 mL) provided by ODFW and NDOW from captured bighorn
304 sheep was collected in EDTA tubes and spun at 4,000 x *g* for 10 minutes to separate the buffy
305 coat. We extracted DNA from this material using a Qiagen DNeasy Blood and Tissue Kit
306 (Qiagen Inc., Valencia, CA, USA). Fecal samples were collected opportunistically while
307 conducting observations of bighorn sheep across the different populations, and were generally a
308 week or less in age, as estimated from pellet color, odor, and surface condition. Fecal samples
309 that were still moist after deposition were dried and then stored at room temperature. Fecal
310 pellets were scraped to target dried epithelial cells on the surface of the pellet (Wehausen, Ramey
311 & Epps, 2004), and we extracted DNA from the scraped material using a modified version of the
312 Aquagenomic Stool and Soil protocol (Multitarget Pharmaceuticals LLC, Colorado Springs, CO;
313 see details in Appendix S4).
314
315 *Genotyping, markers, individual identification, and marker*
316   We used a suite of 16 microsatellite markers in three panels (Table S2) that had
317 previously been used to investigate population connectivity and genetic variability in bighorn
318 sheep (Creech et al., 2017, 2020; Epps, Crowhurst & Nickerson, 2018). Genotyping followed
319 protocols outlined in Epps, Crowhurst & Nickerson (2018). Briefly, all samples were run in at
320 least two (for blood) or three (for feces) independent PCR reactions to generate a consensus

321   genotype for each individual at each locus. For blood samples, any discrepancy between the two
322   replicates resulted in the sample being rerun at that panel, although consistency across replicates
323   was very high. Because allelic dropout can be higher in fecal samples, for those samples a
324   homozygous genotype was considered verified if the single allele was seen in all three replicates.
325   A heterozygous genotype was considered to be verified if each allele was seen in at least two of
326   the three replicates; any other discrepancies resulted in reruns. Other studies on bighorn (e.g.,
327   Epps, Crowhurst & Nickerson, 2018) reported screening for recaptures using as few as 6 loci to
328   achieve a desired probability of identity [PID; Waits, Luikart & Taberlet (2001)] of <0.001 and a
329   probability of identity for full siblings of < 0.05. However, our initial genotyping demonstrated
330   that we needed to genotype at all 16 loci to achieve those thresholds. We identified recaptured
331   individuals using CERVUS (Kalinowski, Taper & Marshall, 2007) by screening for individuals
332   that matched at all 16 loci and removing them from the data set. We repeated this analysis using
333   successively reduced numbers of matching loci and the presence of one to two mismatches (to
334   account for missing data and genotyping error, respectively), until the matches that the program
335   returned seemed unlikely due to geographic location and/or the mismatches were not explainable
336   by simple allelic dropout. Finally, we used GIMLET (Valière, 2002) to calculate two types of error
337   rates in our genotypes: allelic dropout, and the presence of false alleles.

339   *Linkage disequilibrium, Hardy-Weinberg tests, and genetic diversity*
340   We used GENEPOP Version 4.2 on the Web (Rousset, 2008) to conduct the probability test
341   for Hardy-Weinberg equilibrium (HWE) for each population by locus and then for each locus by
342   population, as well as across populations for each locus and across loci for each population
343   (Fisher's method, Fisher 1948). We then used GENEPOP 4.0 Desktop to test for linkage
344   disequilibrium across each pair of loci within each population, and each pair of loci across all
345   populations, applying a sequential Bonferroni correction in both cases across all loci. We used
346   the R package *diveRsity* (Keenan et al., 2013) to calculate population genetic diversity metrics
347   including expected heterozygosity (H_E), observed heterozygosity (H_O), and allelic richness (A_R).
348   We accounted for imbalanced sample sizes amongst populations using rarefaction.

350   *NDVI data*
351   We used 14-day composite, 250 m resolution NDVI data from the Moderate Resolution
352   Imaging Spectroradiometer (eMODIS). We utilized pre-processed data from 2016-2018 obtained
353   from Earth Explorer (https://earthexplorer.usgs.gov/), which is managed by the United States
354   Geological Survey's Earth Resources Observation Center (Jenkerson, Maiersperger & Schmidt,
355   2010).
356   We used GPS data from all collared adult females in each population to generate a single
357   utilization distribution per population for each year from 2016–2018, using the R package
358   *adehabitatHR* (Calenge, 2006). We estimated 95% utilization distributions using the kernel
359   method with the default smoothing parameter (h_{ref}). We did not use least square cross validation
360   (h_{lscv}) due to repeated use of locations, which may cause convergence issues (Kie et al., 2010).

361   We then extracted NDVI data from each population polygon for each 14-day composite
362   image and generated a 90th percentile statistic using program R (R Core Team, 2019). Bighorn
363   sheep are selective feeders; as such, we assume that the 90th percentile NDVI statistic represents
364   a high-quality choice of forage, while accounting for the fact that maximum forage is not always
365   attainable (Creech et al., 2016). Additionally, selecting the 90th percentile excludes the selection
366   of false maximum values caused by measurement error. Finally, we averaged NDVI values
367   across each 14-day composite for the 3-months before and after the first juvenile observed in
368   each population and used this variable as a measure for forage quality for each population pre-
369   and post-parturition.
370
371   *Drivers of juvenile survival*
372   We analyzed our known-fate data in Program MARK (White & Burnham, 1999), to
373   estimate juvenile survival (S) using a Kaplan-Meier estimator (Kaplan & Meier, 1958) with
374   staggered entry (Pollock et al., 1989). First, we considered three population-level measures of
375   exposure to *M. ovipneumoniae* in our models of juvenile survival (Table 1). Primarily, we
376   considered the presence of *M. ovipneumoniae*-infected juveniles in each population, as the
377   limited numbers of adults captured and tested in each population and the lack of annual testing
378   precluded clear estimates of infection rates among adults. However, we also considered whether
379   presence of infected adults (PCR) or exposed adults (cELISA) in each population influenced
380   juvenile survival. Second, we considered two population-level measures of genetic diversity,
381   expected heterozygosity (H_E) and allelic richness (A_R) (Table 1). We used univariate models of
382   the three *M. ovipneumoniae* measures and the two genetic diversity measures as initial screening
383   methods, using Akaike's information criterion, corrected for small sample sizes (AIC_c) to
384   determine which *M. ovipneumoniae* and genetic diversity metric most strongly linked to juvenile
385   survival before proceeding with subsequent analyses.
386   Subsequently, we conducted two analyses of juvenile survival. The first included all
387   study populations (n = 13) and the selected measures of *M. ovipneumoniae* presence in each
388   population, population-level metrics of forage quality (3-month pre- and post-parturition NDVI
389   values) and genetic diversity (expected heterozygosity, H_E) (Table 1), and both additive and
390   multiplicative effects of time. For each model that included the multiplicative effect of time and
391   *M. ovipneumoniae*, we fixed survival interval 1 for the *M. ovipneumoniae* group, as no
392   mortalities occurred during this period. The second analysis included all of the same variables
393   except *M. ovipneumoniae* presence and was restricted to populations where *M. ovipneumoniae*-
394   infected juveniles were not detected (n = 11, see Results). That second analysis was undertaken
395   to determine whether effects of *M. ovipneumoniae* obscured the effect of the other covariates of
396   interest. For both analyses, we used AIC_c and AIC_c weights (w_i) to select the best-supported
397   model. We included a null model in both model selection sets to evaluate model performance
398   (Burnham & Anderson, 2002). We selected the model with the lowest AIC_c and highest w_i as our
399   best-supported model. We used evidence ratios between the top model and competitive models

400   (those within 2 AIC_c units, to evaluate each model relative to the top model (Burnham &
401   Anderson, 2002)).
402
403   *Post-study observations*
404   After our final planned field season in 2018, the single PCR+ adult female in the RSP (of
405   21 tested) died of suspected bluetongue (*Orbivirus* spp.). Because prevalence of PCR+ adult
406   females in this population was low (4.76% of tested - see Results), and no adult males tested
407   PCR+ throughout the study, we considered it possible that no additional PCR+ individuals
408   remained at RSP, potentially removing the source of infection for new juveniles. Therefore, we
409   decided to conduct a single observation of juveniles in the RSP and BHP, populations in early
410   August of 2019; we included BHP given its proximity to RSP. Juveniles in both these
411   populations were typically 4-months old at that time; thus, that observation aligned closely with
412   the 4-month juvenile survival we estimated during the study.
413
414   **Results**
415
416   *Diagnostics*
417   Between 2016 and 2018, 78 adult females were tested via cELISA to determine *M.*
418   *ovipneumoniae* exposure, and 95 adult females were tested via PCR to determine active *M.*
419   *ovipneumoniae* infections. The proportion of adult females PCR tested in each population varied
420   from 0.10 to 0.43 ($\overline{x}$ = 0.27, Table S4). For the 10 adult females in the Santa Rosa
421   metapopulation that were recaptured during our study and retested for infection via PCR, PCR
422   status remained the same for the single positive individual in EML, and the rest were negative.
423   None of the adult females in populations west of U.S. Route 95 (BSP, TCE, TCS, or TCW)
424   showed evidence of *M. ovipneumoniae* exposure (Figure 1, Table S3). However, all of the
425   populations east of U.S. Route 95 (the four populations in the Santa Rosa Mountain's
426   metapopulation, as well as BHP, RSP, TMP, and UOP (Figure 1), except TFK, included adult
427   females with evidence of exposure to *M. ovipneumoniae* (Table S3). We were unable to get a
428   sample from the single individual adult female captured in TFK. The proportion of *M.*
429   *ovipneumoniae* exposed adult females in those eight populations varied between 0.60 in CAL
430   and EML to 1.00 in TMP ($\overline{x}$ = 0.75; Table S4). Only two adult females tested PCR+ to *M.*
431   *ovipneumoniae* infection across all 13 populations (Table S3): one in EML on two occasions
432   (2017 and 2018), and one in RSP in 2016. Additionally, one adult female in BHP yielded an
433   indeterminate PCR test result, meaning *M. ovipneumoniae* detection could not be determined
434   (Besser et al., 2019).
435
436   *Linkage disequilibrium, Hardy-Weinberg tests, and genetic diversity*
437   After removing the fecal samples from recaptured individuals (n = 28), the genetic data
438   set contained 191 individuals at 16 microsatellite loci, representing 10 to 26 ($\overline{x}$ = 15.9)
439   individuals per population (Table S5). The mean rate of false allele occurrence per locus was

440 0.001 and the mean allelic dropout rate across all loci was 0.008 (range = 0.000-0.017). No locus
441 was determined to be out of Hardy-Weinberg equilibrium by either test employed (Tables S6 &
442 S7). Evaluating each locus pair by population showed no evidence of linkage disequilibrium
443 ($p_{critical}$ = 0.000035 for α = 0.05); evaluating linkage for each pair of loci across populations using
444 Fisher's test indicated that BL4 and HH62 were in disequilibrium (p = 0.00039; $p_{critical}$ = 0.00042
445 for α = 0.05). However, those loci have not appeared to be in disequilibrium in other bighorn
446 sheep studies (e.g., Epps, Crowhurst & Nickerson, 2018), suggesting that this relationship may
447 have been an artifact. Thus, and because linkage disequilibrium is more likely to bias estimates
448 of genetic structure rather than estimates of genetic diversity as employed in this study, we
449 retained all 16 loci in our analyses. H_E across the 13 study populations varied from 0.26 in BSP
450 to 0.48 in EML ($\overline{x}$ = 0.37; Table S5) and A_R varied from 1.82 in BSP to 2.88 in SAW ($\overline{x}$ = 2.37;
451 Table S5).

452

453 *NDVI data*
454   Pre- and post-parturition NDVI varied spatially and temporally (Table S8). BSP had
455 consistently low pre-parturition NDVI, whilst EML and TMP had consistently high pre-
456 parturition NDVI (Table S8). Post-parturition NDVI was lowest in BHP in 2018, was
457 consistently low in RSP but was consistently high in TCE and EML (Table S8). Pre- and post-
458 parturition NDVI values were not correlated (Table S9).

459

460 *Pregnancy rates and observation of juvenile bighorn sheep*
461   Seventy-six of the 82 (93%), pregnancy tests conducted across all 13 populations
462 between 2016 and 2018 were positive (Table S10). Six of the 82 tests conducted were on
463 recaptured adult females from EML (n = 3) and SAW (n=3), all of which were positive. All
464 populations had 100% pregnancy rates, except for TCE (67%; n = 8/12), TCS (50%; n = 1/2),
465 and TFK (0%; n = 0/1) in 2016. The single collared adult female in TFK did not yield a positive
466 pregnancy test result in January, but was later observed in early June with a juvenile, suggesting
467 that the test was administered too early to detect a positive pregnancy result. Sixty-five of those
468 82 (79%) pregnant adult females survived to parturition; of those 59 (91%) were observed with
469 juveniles. Population pregnancy rates were not correlated with genetic diversity (Pearson
470 pairwise correlation; H_E, r = 0.24, p = 0.272; A_R, r = 0.14, p = 0.529).
471   We observed 121 juveniles with radio-collared adult females between 2016 and 2018;
472 78% of radio-collared adult females were observed with juveniles. Populations with the lowest
473 proportion of radio-collared adult females with juveniles were BSP (2016 – 0.00; 2017 – 0.67;
474 2018 – 0.33), and TCE (2016 – 0.64; 2017 – 0.64; 2018 – 0.17), although sample sizes were
475 small in BSP (n = 3). A set of twins was observed with a radio-collared adult female in both the
476 BHP and MCK populations in 2018.
477   The observation rate of collared adult females with juveniles varied from 50 to 100%,
478 with a mean observation rate of 94% across all semi-monthly sampling periods and study
479 populations (Table S11). Throughout the study, we collected samples suitable for *M*.

480 *ovipneumoniae* testing from 17 juvenile mortalities (Table S12). All juvenile mortalities (n = 15)
481 recovered from the BHP (n = 1) and RSP (n = 14) populations tested positive for *M.*
482 *ovipneumoniae*. None of the other juvenile mortalities from BSP (n = 1) or TCS (n = 1) tested
483 positive for *M. ovipneumoniae*. The *M. ovipneumoniae* strain-type of the samples collected from
484 the BHP and RSP juveniles matched the strain-type (NV_BHS_SantaRosas_2651_2014_4;
485 Kamath et al., 2019) of all the other previously strain-typed bighorn sheep at the available
486 sequences from the Santa Rosa meta-population and Rattlesnake population (Table S13).
487
488 *Drivers of juvenile survival*
489   Preliminary analyses with univariate models of the relationship between juvenile survival
490 and different population-level measures of *M. ovipneumoniae* revealed that the presence of *M.*
491 *ovipneumoniae*-infected juveniles in the population was a much stronger predictor of juvenile
492 survival than the presence of infected (PCR+) or exposed (ELISA+) adults (Table S14).
493 Preliminary analyses with univariate models of juvenile survival as a function of genetic
494 diversity demonstrated that H_E was a stronger predictor of juvenile survival than A_R (Table S14).
495 Therefore, in our subsequent multivariate models of juvenile survival, we used the presence of
496 *M. ovipneumoniae*-infected juveniles in each population as our measure of *M. ovipneumoniae*
497 presence, and H_E as our measure of genetic diversity. In those multivariate analyses, we
498 identified two competing models predicting survival probability of juveniles across all
499 populations (Table 2). Both models contained only two predictors: 1) whether *M.*
500 *ovipneumoniae*-infected juveniles were detected, and 2) time. The model containing time as a
501 multiplicative effect with *M. ovipneumoniae* had 2.36 times more support than the competing
502 model that treated time as an additive effect (Table 2), meaning that the temporal pattern of
503 juvenile mortality differed in populations where *M. ovipneumoniae*-infected juveniles were
504 present. Neither forage quality (pre- and post-parturition NDVI), nor genetic diversity (H_E)
505 predicted survival probability of juveniles (Table 2).
506   The odds of a juveniles surviving to 4-months of age in our study populations where *M.*
507 *ovipneumoniae*-infected juveniles were detected were 8.00 (95% CI: -4.00–255.78) times less
508 likely than juveniles in populations where we did not detect *M. ovipneumoniae*-infected juveniles
509 (Table 3). The derived probability of survival for the entire 4-month annual study period for
510 juveniles in populations where *M. ovipneumoniae*-infected juveniles were detected was 0.02
511 (95% CI: 0.00–0.13) compared to 0.44 (95% CI: 0.29–0.59) for juveniles in other populations
512 (Figure 2B).
513   We observed no significant difference in semi-monthly juvenile survival probability
514 between populations where *M. ovipneumoniae*-infected juveniles were not detected in
515 observations at 0.5, 1, and 1.5-months post-parturition (Figure 2A). However, semi-monthly
516 survival probability in populations where *M. ovipneumoniae*-infected juveniles were present
517 were significantly lower 2-months (*S* exposed = 0.42, 95% CI: 0.24–0.62; *S* unexposed = 0.88,
518 95% CI: 0.75–0.95) and 2.5-months (*S* exposed = 0.40, 95% CI: 0.16–0.70; *S* unexposed = 0.94,
519 95% CI: 0.82–0.98) post-parturition (Figure 2A). At 3, 3.5, and 4-months post-parturition,

520 estimates in semi-monthly survival probability again did not differ between populations where
521 *M. ovipneumoniae*-infected juveniles were or were not detected (Figure 2A). The increase in
522 uncertainty in our estimates is tied to decreasing sample size as individuals died.
523 Although no direct effect of either population genetic diversity or post-parturition NDVI
524 was supported based on our model selection approach, both variables were highly correlated with
525 the presence of *M. ovipneumoniae* (H_E = 0.58; post-NDVI = -0.66, Table S9). In fact, the two
526 populations where *M. ovipneumoniae*-infected juveniles were present, BHP and RSP, had
527 relatively high genetic diversity (H_E: BHP = 0.43; RSP = 0.45) compared to other study
528 populations (Table S5). Similarly, RSP had low post-parturition NDVI values relative to the
529 other populations in 2017 and 2018, while the same was the case for BHP in 2018 (Table S8).
530 In the second analysis, which excluded the two populations where *M. ovipneumoniae*-
531 infected juveniles were present, competing models included both indices of forage quality and
532 population genetic diversity as predictors of juvenile survival (Table 2), as well as a time-varying
533 effect. However, the 95% confidence intervals for the parameter estimates of the nutritional and
534 population genetic diversity model both overlapped zero, and the null model with no predictors
535 was 2nd ranked. The top competing model, a time-varying model, had 1.69 times more support
536 than the next best model, the null model (Table 2).
537
538 *Post-study observations*
539 In early August of 2019, following the death of the single adult female in RSP that tested
540 PCR+ for *M. ovipneumoniae*, we located the two remaining collared individuals in the BHP
541 (linked to RSP by adult male and female movements, data not shown). Neither the collared adult
542 females nor any of the eight other adult females we observed in this population had juveniles
543 (0% juvenile survival). We then located 14 of the RSP collared adult females, of which 11 had
544 juveniles (~79% juvenile survival). We located approximately 35 adult females total in the RSP
545 population and observed approximately 27 juveniles, which equates to an approximate 4-month
546 survival rate of ~77%.
547
548 **Discussion**
549 We found that *M. ovipneumoniae* had a strong negative effect on juvenile survival in
550 populations where infected juveniles were detected. Juveniles in those populations had a 4-
551 month survival probability more than 20 times lower than juveniles in populations where we did
552 not detect infected juveniles. Indeed, in one affected population (RSP), only two juveniles out of
553 40 over three years survived. Presence of adult bighorn sheep with evidence of past exposure to
554 *M. ovipneumoniae*, and in one population the presence of an adult infected at time of capture,
555 were not strong predictors of juvenile survival. Moreover, juvenile survival was not detectably
556 influenced by forage quality or population genetic diversity, even when excluding populations
557 where *M. ovipneumoniae*-related juvenile mortality was observed. Strikingly, the death of a
558 single infected adult female in a heavily impacted population appeared to drive an immediate

559 recovery of juvenile survival in that population in the subsequent year, underscoring the
560 potentially pivotal role of chronically infected adult females in outbreaks of this disease.
561   In our study, four-month juvenile survival probability in populations where infected
562 juveniles were present (0.02; 95% CI: 0.00–0.13) were comparable to some of the lowest
563 observed juvenile survival probabilities reported in other studies where pneumonia epizootics
564 were present. In Rocky Mountain bighorn sheep in the Hells Canyon system of Idaho, Oregon,
565 and Washington, survival to weaning at 4–5 months of age had a median value of 0.10, but
566 ranged from <0.01–0.69, with peak mortality occurring between 1.5 and 2.5 months of age
567 (Cassirer et al., 2013). In the Black Hills of South Dakota, Smith et al. (2014) reported 52-week
568 survival to be only 0.02 (95% CI: 0.01–0.07), with the majority of juveniles (~75%) dying within
569 the first 2 months after parturition. We observed similar temporal patterns to those studies, with
570 most mortality related to disease in our study occurring between 1.5 and 3-months after
571 parturition. In the Mojave Desert of California, in a 3-year study of desert bighorn sheep
572 following an outbreak of *M. ovipneumoniae*, the proportion of juveniles surviving varied from
573 0.00 to 0.92 in populations where at least five juveniles were monitored, and survival in most
574 populations improved in later years (Dekelaita 2020). Unlike Cassirer et al. (2013) and Smith et
575 al. (2014), we did not address the effect of predators on juvenile survival. However, it should be
576 noted that even if cause-specific mortality were to be assigned to a predator, the ultimate cause
577 of mortality might still be *M. ovipneumoniae*. Although *M. ovipneumoniae* is considered the
578 primary agent of pneumonia, other diseases, and the potential for co-infection effects may be
579 important, but we did not assess them. Periods of low juvenile survival due to pneumonia can
580 result in demographic consequences such as declining population growth rates (Manlove et al.,
581 2016) and even skewed population structures (Festa-Bianchet, Gaillard & Côté, 2003).
582   Our study adds to the evidence that dynamics of respiratory pneumonia in bighorn sheep
583 may be strongly influenced by the presence of only a small number of infected adults. We
584 detected only one infected female adult bighorn sheep across two populations where *M.
585 ovipneumoniae*-infected juvenile bighorn sheep were observed. However, the presence of
586 infected adults at capture was not the strongest predictor of juvenile survival. For instance, in
587 EML, an infected female adult was present in 2017; she was still infected when retested in 2018.
588 Despite that, one out of four juveniles born in 2017 and five of six juveniles born in 2018
589 survived. It is unclear why the presence of this apparently chronically-infected female did not
590 lead to juvenile mortality in this case, although Plowright et al., (2017) noted that juvenile
591 survival was not directly related to the mother's infection status in the study. The lack of juvenile
592 die-off could be explained by other factors, e.g., the stochastic nature of exposure and the
593 possibility of co-infection. We attempted to capture other factors that may play a role, e.g.,
594 measures of forage quality and population genetic diversity, but we did not detect any direct
595 effect of those factors. Additionally, we were unable to assess the interaction of these variables
596 with *M. ovipneumoniae* infection because our measures of those factors were at the population
597 level. Conversely, juvenile survival in RSP was extremely poor from 2016–2018, where a single
598 infected adult female was detected in 2016. Although we did not retest her and therefore do not

599   know whether she was chronically infected, after her death in late 2018, juvenile survival

600   rebounded sharply in 2019. In a satellite population discovered in the course of this study, BHP,

601   one female out of three captured in 2018 yielded an indeterminate PCR result. We surmise that

602   additional captures likely would have detected infected adults given that infected juveniles were

603   detected in 2018 and no surviving juveniles were detected in either 2018 or 2019. Experimental

604   removals of chronically-infected adult females in other bighorn sheep studies (e.g., Garwood et

605   al., 2020) demonstrate that chronically-infected adult females can strongly influence juvenile

606   survival when prevalence is low.

607   We did not detect influences of population genetic diversity on juvenile survival,

608   although we observed low to very low population genetic diversity throughout our study area,

609   with H_E ranging from 0.26–0.48 (Table S5). In contrast, H_E values derived for 11 native desert

610   bighorn sheep populations in the Mojave Desert of California, using 15 of the 16 microsatellite

611   loci we employed, ranged from 0.50–0.69 (Epps, Crowhurst & Nickerson, 2018). However, our

612   study design did not account for possible interactions between these variables and presence of *M.*

613   *ovipneumoniae*-infected juveniles, because *M. ovipneumoniae*-related juvenile mortalities were

614   confirmed in only two of 13 populations. Our estimates of genetic diversity were also only

615   derived from 16 microsatellite loci, most putatively neutral; estimates based on more markers

616   across the genome or additional genes associated with immune function could have been more

617   informative. Interestingly, the populations where *M. ovipneumoniae*-related juvenile mortality

618   was confirmed had relatively high genetic diversity (Table S5). In fact, the higher genetic

619   diversity observed at RSP likely resulted from gene flow from males dispersing from the Santa

620   Rosa metapopulation (NDOW, unpublished data). Such movements also appeared to have spread

621   this strain of *M. ovipneumoniae* from the Santa Rosa metapopulation to BHP and RSP; indeed,

622   only one strain is known to occur throughout the populations where we detected infected

623   individuals (Table S13). Genetic diversity in populations to the west of U.S. Route 95, in

624   particular, was strikingly low (H_E was 0.26–0.33) due to founder effects from translocation

625   history (see above). Genetic diversity observed by Olson, Whittaker & Rhodes (2013) for the

626   source population, Hart Mountain, was noticeably higher ($H_E = 0.42$). We note that the

627   pregnancy rate in one of those populations (TCE) likewise was strikingly low (67%, n = 8/12) in

628   2016. Although the adult females that we had the opportunity to test in 2017 and 2018 (n = 3

629   total) were pregnant, the numbers of adult females observed with juveniles in 2017 and 2018

630   were equal to or lower than 2016. Yet, those populations were apparently free of *M.*

631   *ovipneumoniae* during our study. Such low pregnancy rates suggest that inbreeding or low

632   genetic diversity could influence performance of those populations (Olson, Whittaker & Rhodes,

633   2012), although we cannot rule out other factors. These contrasts highlight the challenges of

634   maintaining genetic diversity while limiting spread of disease, particularly in reintroduced

635   systems of populations where compounding founder effects have resulted in some of the lowest

636   reported values for genetic diversity in wild bighorn sheep populations.

637   Forage quality as assessed by NDVI values pre- and post-parturition likewise did not

638   appear to influence juvenile survival consistently across the study. Again, our ability to evaluate

639   how variation in forage quality influenced survival when *M. ovipneumoniae* was present was
640   limited. We note, however, that of the three populations where *M. ovipneumoniae*-infected adult
641   females or juveniles were detected, juvenile survival was lowest where forage quality was lowest
642   (BHP and RSP, Table S8, average juvenile survival = 0 and 0.07, respectively). In contrast, EML
643   had the highest or second-highest pre- and post- parturition NDVI values (Table S8) and higher
644   juvenile survival ($\overline{x}$ = 0.54; range = 0.25–0.83). Taken together, these anecdotal patterns suggest
645   that the link between forage quality and *M. ovipneumoniae* dynamics within infected populations
646   bears further investigation.
647   Our study demonstrated that reintroduced populations of "California"-managed bighorn
648   sheep in southeastern Oregon and northern Nevada have been strongly influenced by respiratory
649   disease, but not all populations are currently affected. Where infected juveniles were present,
650   rates of juvenile mortality that we observed would lead to a dramatic decline in populations if
651   those patterns persisted, as observed by (Manlove et al., 2016). However, the rapid improvement
652   in juvenile survival in one badly-affected population following the death of the single adult
653   female known to be infected with *M. ovipneumoniae* at the start of the study suggests that
654   identification and removal of chronically-infected adult females could be a successful
655   management strategy in this system, as tested for a Rocky Mountain bighorn sheep population by
656   Garwood et al. (2020). The very low estimates of genetic diversity we observed in some
657   populations suggest that actions to manage for improved genetic diversity may be warranted,
658   although natural movements between several study populations simultaneously resulted in
659   increased genetic diversity and spread of *M. ovipneumoniae*. Thus, any genetic management
660   strategy employed would have to mitigate the potential impact on spread of disease.
661
662   **Conclusions**
663   We found that populations with *M. ovipneumoniae*-infected juveniles had significantly
664   lower juvenile survival, while no direct effect of either genetic diversity or pre- and post-
665   parturition forage quality on juvenile survival was detected. Presence of *M. ovipneumoniae*-
666   infected or exposed adults was less predictive of juvenile survival. Our findings add to the body
667   of evidence that *M. ovipneumoniae* can have deleterious effects on juvenile survival across the
668   various bighorn sheep lineages and the different ecosystems they inhabit. After the mortality of
669   the only PCR+ adult female that we detected in one of the populations most affected by
670   respiratory disease during the first three years of our study, we found increased juvenile survival
671   and detected no infected juveniles in the following year. This natural experiment suggests that in
672   populations where prevalence of PCR+ adults is low, removal of infected adults or adults
673   identified as chronic carriers of *M. ovipneumoniae*. However, our ability to detect those
674   influences may have been limited by small number of populations where respiratory disease was
675   observed and high correlations between genetic diversity and presence of disease resulting from
676   translocation history and patterns of connectivity in this system. Future studies should evaluate
677   the interaction of genetic diversity and disease in systems with larger numbers of populations
678   affected by respiratory disease.

679

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891

Figure 1

Cumulative summer utilization distributions of adult female bighorn sheep populations considered in this study.

Cumulative summer utilization distributions of adult female bighorn sheep populations considered in this study. Overlapping polygons indicate shared habitat. Solid lined polygons, all to the west of U.S. Route 95, indicate populations unexposed to *Mycoplasma ovipneumoniae*, with dashed lines indicating exposed populations to the east of U.S. Route 95. Line fill indicates populations, EML and RSP, where a single adult female *M. ovipneumoniae* infection was detected. Green colored polygons indicate populations, BHP and RSP, where dead juveniles infected with *M. ovipneumoniae* were detected. Populations include – Bowden Hills (BHP), Blue Mountain (BSP), Calicos (CAL), Eight Mile (EML), Martin Creek (MCK), Rattlesnake (RSP), Sawtooth (SAW), Trout Creeks – east (TCE), Trout Creeks – south (TCS), Trout Creeks – west (TCW), Three Forks (TFK), Ten Mile (TMP) and Upper Owyhee (UOP).

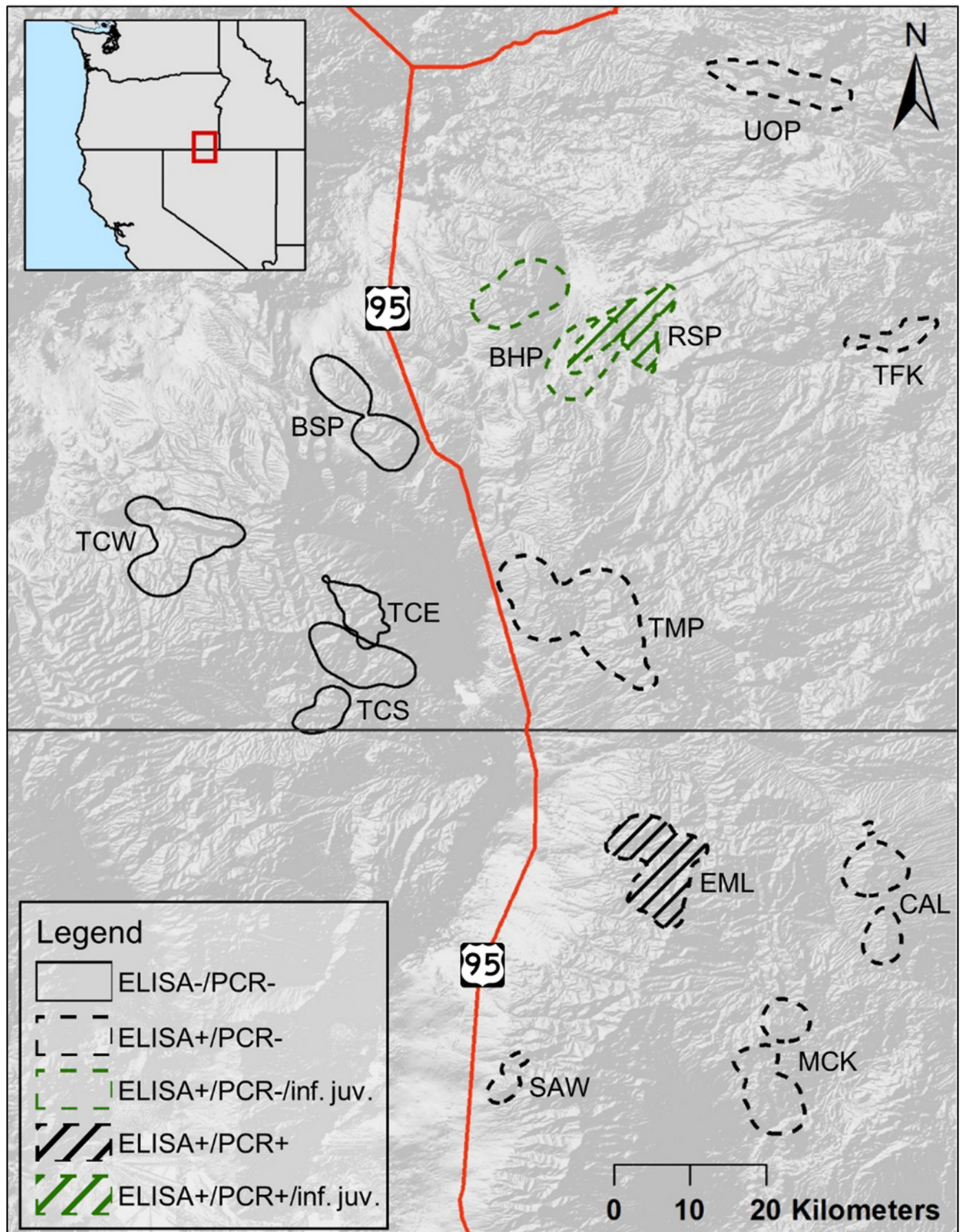
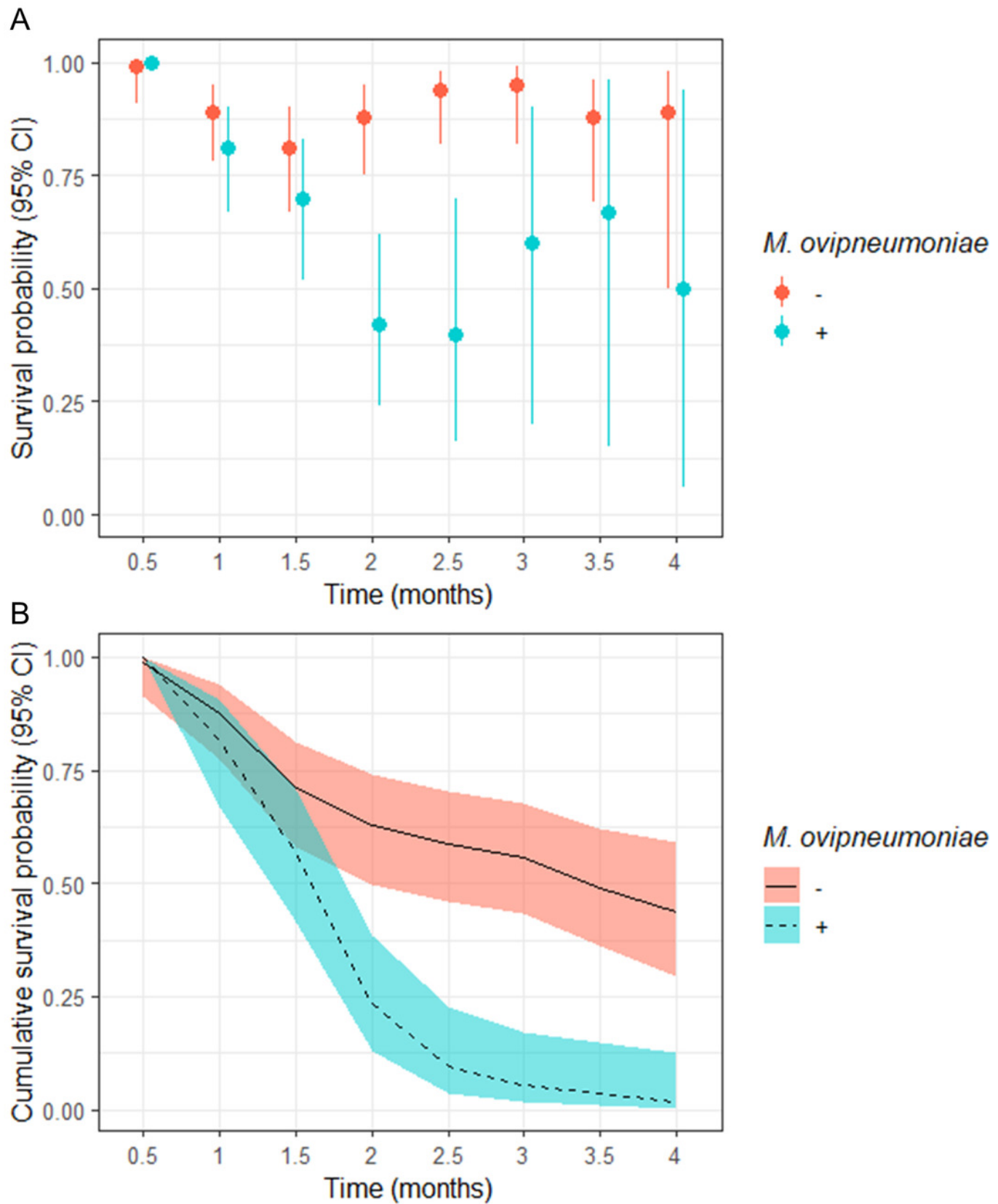



Figure 2

'(A) Semi-monthly survival probabilities and (B) 4-month cumulative survival probabilities of juvenile bighorn sheep (*Ovis canadensis*) in populations across southeastern Oregon and northern Nevada.

'(A) Semi-monthly survival probabilities and (B) 4-month cumulative survival probabilities of juvenile bighorn sheep (*Ovis canadensis*) estimated using a Kaplan-Meier estimator with staggered entry and known-fate data collected in 13 populations in southeastern Oregon and northern Nevada. Survival probabilities and 95% confidence intervals are for populations where *Mycoplasma ovipneumoniae* was and was not detected.

Description of variables considered in known-fate models predicting survival of juvenile bighorn sheep (*Ovis canadensis*) in populations across southeastern Oregon and northern Nevada.

Description of variables considered in known-fate models predicting survival of juvenile bighorn sheep (*Ovis canadensis*) in populations across southeastern Oregon and northern Nevada. All statistical measures were considered at the population level.

Measure	Category	Measure type	Statistical measure (range)
. (null)		Intercept only model	
time	Temporal	Time-varying	
T	Temporal	Linear time trend	
M. ovipneumoniae status	Bacteria	*M. ovipneumoniae* status (+/-), as determined by presence of infected juveniles.	Binary
M. ovipneumoniae status	Bacteria	*M. ovipneumoniae* status (+/-) as determined by presence of infected (PCR+) adults	Binary
M. ovipneumoniae status	Bacteria	*M. ovipneumoniae* status (+/-) as determined by presence of infected (cELISA+) adults	Binary
A_R (Allelic richness)	Genetic	Measure of genetic diversity	Continuous (values btw. 1.82–2.88)
H_E (Expected heterozygosity)	Genetic	Measure of genetic diversity	Continuous (values btw. 0–1)
pre-NDVI (Pre-parturition NDVI)	Nutrition	90th percentile 3-month pre-parturition mean NDVI	Continuous (values btw. -0.2–1)
post-NDVI (Post-parturition NDVI)	Nutrition	90th percentile 3-month post parturition mean NDVI	Continuous (values btw. -0.2–1)

Model selection results for known fate models predicting cumulative 4-month survival of juvenile bighorn sheep (*Ovis canadensis*) in southeastern Oregon and northern Nevada for the period 2016–2018.

Model selection results for known fate models predicting cumulative 4-month survival of juvenile bighorn sheep (*Ovis canadensis*) in southeastern Oregon and northern Nevada for the period 2016–2018. For analysis 1, we included a binary group variable for populations where *Mycoplasma ovipneumoniae* was (n = 2) and was not detected (n = 11). We modeled time as a constant (.), time-varying (time), linear (T), and random effect. Covariates modeled include expected heterozygosity (H_E), and pre- and post-parturition NDVI. For analysis 2, we only modeled populations (n = 11) with no observed *M. ovipneumoniae* mortalities. We modeled time as a constant (.), time-varying (time), and linear (T) effect. We included the same covariates in data set 2 that we used in data set 1.

Analysis	Model	K	AIC_c	ΔAIC_c	w_i	ML
1	*M. ovipneumoniae* $\times$ time	15*	366.51	0.00	0.30	1.00
	M. ovipneumoniae + time	9	368.23	1.71	0.13	0.42
	M. ovipneumoniae $\times$ time + H_E	16*	368.58	2.06	0.11	0.36
	M. ovipneumoniae $\times$ time + post-NDVI	16*	368.63	2.12	0.10	0.35
	M. ovipneumoniae $\times$ time + pre-NDVI	16*	368.64	2.12	0.10	0.35
	M. ovipneumoniae + time + H_E	10	370.02	3.51	0.05	0.17
	M. ovipneumoniae + time + pre-NDVI	10	370.23	3.72	0.05	0.16
	M. ovipneumoniae + time + post-NDVI	10	370.31	3.79	0.04	0.15
	M. ovipneumoniae $\times$ time + H_E + post-NDVI	17*	370.66	4.15	0.04	0.13
	M. ovipneumoniae $\times$ time + H_E + pre-NDVI	17*	370.69	4.18	0.04	0.12
	M. ovipneumoniae $\times$ time + pre-NDVI + post-NDVI	17*	370.77	4.25	0.04	0.12
	M. ovipneumoniae $\times$ time + H_E + pre-NDVI + post-NDVI	18*	372.80	6.29	0.01	0.04
	time + post-NDVI	9	383.02	16.51	0.00	0.00
	time + H_E	9	387.94	21.42	0.00	0.00
	Time	8	392.21	25.70	0.00	0.00
	time + pre-NDVI	9	394.22	27.71	0.00	0.00
	M. ovipneumoniae	2	400.46	33.94	0.00	0.00
	post-NDVI	2	412.59	46.08	0.00	0.00
	H_E	2	417.59	51.07	0.00	0.00
	T	2	418.76	52.24	0.00	0.00

	. (null)	1	422.68	56.17	0.00	0.00
	pre-NDVI	2	424.41	57.90	0.00	0.00
2	Time	8	210.79	0.00	0.23	1.00
	. (null)	1	211.84	1.05	0.14	0.59
	time + pre-NDVI	9	212.52	1.73	0.10	0.42
	time + H_E	9	212.70	1.91	0.09	0.38
	time + post-NDVI	9	212.78	1.99	0.09	0.37
	T	2	213.27	2.48	0.07	0.29
	post-NDVI	2	213.60	2.82	0.06	0.24
	pre-NDVI	2	213.67	2.88	0.05	0.24
	H_E	2	213.69	2.90	0.05	0.23
	time + H_E + pre-NDVI	10	214.19	3.41	0.04	0.18
	time + pre-NDVI + post-NDVI	10	214.41	3.62	0.04	0.16
	time + H_E + post-NDVI	10	214.78	4.00	0.03	0.14
	time + H_E + pre-NDVI + post-NDVI	11	216.25	5.46	0.02	0.07

1 *Models where survival interval 1 for the *M. ovipneumoniae* group were fixed as no mortalities occurred during that interval

The outputs from the top models in analysis 1 (*M. ovipneumoniae* x time) and analysis 2 (null) predicting survival of juvenile bighorn sheep (*Ovis canadensis*) in southeastern Oregon and northern Nevada.

The outputs from the top models in analysis 1 (*M. ovipneumoniae* x time) and analysis 2 (null) predicting survival of juvenile bighorn sheep (*Ovis canadensis*) in southeastern Oregon and northern Nevada. Analysis 1 includes all study populations, while analysis 2 only includes populations (n = 11) where *Mycoplasma ovipneumoniae* was not detected.

Analysis	Covariate	Effect on survival	Odds-ratio	Estimate	SE	95% CI Lower	95% CI Upper
1	Intercept			2.08	1.06	0.00	4.16
	M. ovipneumoniae	↓	8.00	-2.08	1.77	-5.54	1.39
	time 1	↑	9.50	2.25	1.46	-0.61	5.12
	time 2	↑	1.00	0.00	1.13	-2.22	2.22
	time 3	↓	1.89	-0.64	1.12	-2.84	1.56
	time 4	↓	1.05	-0.05	1.16	-2.33	2.23
	time 5	↑	1.83	0.61	1.22	-1.78	2.99
	time 6	↑	2.44	0.89	1.28	-1.63	3.41
	time 7	↓	1.09	-0.09	1.23	-2.49	2.32
	M. ovipneumoniae × time						
	1*	↑	1.00	0.00	0.00	0.00	0.00
	M. ovipneumoniae × time 2	↑	4.37	1.48	1.85	-2.16	5.11
	M. ovipneumoniae × time 3	↑	4.36	1.47	1.85	-2.15	5.09
	M. ovipneumoniae × time 4	↓	1.33	-0.29	1.88	-3.96	3.39
	M. ovipneumoniae × time 5	↓	2.75	-1.01	1.97	-4.88	2.86
	M. ovipneumoniae × time 6	↓	1.63	-0.49	2.12	-4.64	3.66
	M. ovipneumoniae × time 7	↑	2.18	0.78	2.24	-3.60	5.16
2	Intercept			2.08	1.06	0.00	4.16
	time 1	↑	9.50	2.25	1.46	-0.61	5.12
	time 2	↓	1.00	0.00	1.13	-2.22	2.22

time 3	↓	1.89	-0.64	1.12	-2.84	1.56
time 4	↓	1.05	-0.05	1.16	-2.33	2.23
time 5	↑	1.83	0.61	1.22	-1.78	2.99
time 6	↑	2.44	0.89	1.28	-1.63	3.41
time 7	↓	1.09	-0.09	1.23	-2.49	2.32

1 *M. ovipneumoniae* x time 1 is fixed as no mortalities were observed during this interval.