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LETTER

Cascading effects of a disease outbreak in a remote protected area

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Abstract

Disease outbreaks induced by humans increasingly threaten wildlife communities worldwide. Like predators, pathogens can be key top-down forces in ecosystems, initiating trophic cascades that may alter food webs. An outbreak of mange in a remote Andean protected area caused a dramatic population decline in a mammalian herbivore (the vicuña), creating conditions to test the cascading effects of disease on the ecological community. By comparing a suite of ecological measurements to pre-disease baseline records, we demonstrate that mange restructured tightly linked trophic interactions previously driven by a mammalian predator (the puma). Following the mange outbreak, scavenger (Andean condor) occurrence in the ecosystem declined sharply and plant biomass and cover increased dramatically in predation refuges where herbivory was historically concentrated. The evidence shows that a disease-induced trophic cascade, mediated by vicuña density, could supplant the predator-induced trophic cascade, mediated by vicuña behaviour, thereby transforming the Andean ecosystem.

KEYWORDS
disease, high Andes, Puma concolor, Sarcoptic mange, Trophic cascades, Vicugna vicugna, Vultur gryphus

Julia D. Monk and Justine A. Smith contributed equally

INTRODUCTION

Disease outbreaks are a growing threat to animal communities worldwide. The consequences of human activities, including habitat modification (North et al., 2015), climate change (Cohen et al., 2020) and animal rearing (Hernández et al., 2019) contribute to disease emergence in wild animal populations. Anthropogenic acceleration of disease spread in wildlife (e.g. Price et al., 2016) can have cascading effects on ecological communities (Buck & Ripple, 2017; Stapp, 2007; Wilmers et al., 2006). But unlike endemic top-down forces from native predators or pathogens, which often have long evolutionary histories with prey and hosts (Burak et al., 2018; Mitchell, 2009; Sih, 1984), novel disease outbreaks can quickly reduce animal numbers before behavioural responses evolve (Holdo et al., 2009; Martin et al., 2018). Such rapid outbreaks create the potential for dramatic disruptions to community dynamics and ecosystem functioning.

Nevertheless, few trophic cascade studies investigate the knock-on effects of diseases throughout food webs (Buck & Ripple, 2017). Trophic cascades occur when top-down forces control herbivore density or behaviour, thereby reducing herbivory and increasing plant abundance; while predators, pathogens, and parasites can all theoretically induce trophic cascades, the indirect effects of predators have received far greater study (Buck & Ripple, 2017). As environmental change increases the frequency and intensity of disease outbreaks in wildlife globally (Daszak et al., 2000; Harvell et al., 2002), elucidating the ecological consequences of disease has urgent bearing on conservation.

Disease outbreaks can often accompany multiple stressors, making it difficult to isolate ecosystem-wide impacts. Consequently, reliably attributing ecological changes to a novel disease outbreak is possible only when substantive ecological data exist as a baseline for comparison (e.g. Holdo et al., 2009; Schultz et al., 2016). The unpredictability of these outbreaks thus makes it challenging to examine the cascading effects of disease independently from other biotic interactions and anthropogenic disturbances. Furthermore, even when cascading disease effects have been examined, almost no studies have quantified how pathogens disrupt existing top-down forces on the host species (Buck & Ripple, 2017, but see Lafferty, 2004). We report here on the effects of an outbreak of Sarcoptic mange (*Sarcoptes scabiei*) in an Andean protected area where long-term research has examined trophic cascades involving pumas (*Puma concolor*, Linnaeus 1771), vicuñas (*Vicugna vicugna*, Molina 1782), condors (*Vultur gryphus*, Linnaeus 1758) and plant communities (Donadio & Buskirk, 2016; Donadio et al., 2010, 2012; Perrig et al., 2016; Smith et al., 2019a,b). Using these baseline data, we were able to assess the community-wide consequences of disease outbreak in an otherwise undisturbed remote protected area.

Mange is a virulent skin parasite that can cause high mortality and symptoms including lesions, alopecia, hyperkeratosis, infection, and a weakened immune system (Bornstein et al., 2008). Mange is expanding to new species and ecosystems (Astorga et al., 2018; Escobar et al., 2021) and outbreak frequency is likely amplified as wild mammals come into increasing contact with domestic species (Niedringhaus et al., 2019). Indeed, contact with domestic animals is one of the two primary causes of disease emergence in wildlife (Tompkins et al., 2015). Here, a mange outbreak emerged in vicuñas in San Guillermo National Park (SGNP), Argentina, from uncertain origins, though it is speculated that it spread to wild camelids in the region from llamas (*Lama glama*, Linnaeus 1758) that were released in an adjacent reserve (Ferreyra et al., 2022).

In SGNP, a top carnivore, the puma, preys mostly on the vicuña (Donadio et al., 2012), which grazes on only a few plant species (the park's dominant grasses, rushes, and sedges; Cajal, 1989). Puma kill rates are high (Smith et al., 2020), providing an important dietary subsidy to an avian scavenger, the Andean condor. Puma-predated vicuña carcasses account for 88% of the local condor diet (Perrig et al., 2016), and although condors also feed on livestock outside the park, high food availability tethers a large number of otherwise widely ranging condors to SGNP (Perrig et al., 2020). Previous work has found that the impacts of vicuña herbivory are highly concentrated in the park's vast open plains, where vicuñas are relatively safe from puma predation (Donadio & Buskirk, 2016; Smith et al., 2019a). Exclosure experiments revealed that vicuña grazing in plains reduced vegetation biomass by 85% and cover by over 50% (Donadio & Buskirk, 2016). In contrast, vicuña impacts on vegetation are minimal in the high-risk canyons, which have similar vegetation to plains but where vicuñas are highly vigilant. Though vicuñas are drawn to meadows during the day despite high predation risk due to the abundance of highly nutritious forage, they are also highly vigilant in these habitats, avoiding them at dangerous hours, and ample water and nutrients allow meadows to recover quickly from herbivory (Donadio & Buskirk, 2016; Smith et al., 2019b). Thus, pumas trigger behavioural trade-offs by vicuñas that result in clear vegetation patterning, concentrating the impacts of herbivory in plains (Donadio & Buskirk, 2016).

This well-characterised portrait of a simple vertebrate food web allowed us to evaluate the community-wide effects of a catastrophic mange outbreak in vicuñas in 2015. We hypothesised that disease would fundamentally restructure the tightly linked trophic relationships previously controlled by puma predation and associated risk. First, we verified that mange, rather than increased predation, was the cause of the vicuña decline by monitoring vicuña densities and disease occurrence, vicuña and puma spatial distributions, and puma predation behaviour. Second, we

predicted that condors would eventually reduce their activity in SGNP without steady carcass provisioning by pumas, and used GPS data from tagged condors to test this prediction. Finally, we predicted that mange would induce a trophic cascade in plains where vicuña herbivory was high, but not in habitats where predation risk and bottom-up controls had previously limited vicuña impacts on vegetation. We used both field measurements of vegetation and remote sensing to test for this habitat-dependent, disease-induced trophic cascade. Using multiple lines of evidence across trophic levels, we trace the cascading impacts of a novel disease outbreak on a pristine, high Andean ecosystem. We provide clear evidence that disease is a non-equivalent top-down force to predation, controlling herbivore density rather than behaviour with fundamentally different outcomes across trophic levels.

MATERIALS AND METHODS

Study system

SGNP is a 166,000 ha reserve in the central Andes on the western border of Argentina (29°14'S, 69°21'W; 2000–5600 m elevation; precipitation per year <300 mm). Camelids are the only large mammalian herbivores, and the park contains the world's southernmost population of vicuñas. The park features three main habitat types: plains, canyons, and meadows, which we classified using a random forest classifier with field-verified habitat locations (see Supplementary Material). Vast, open plains with sparse grasses and shrubs make up nearly half (48.9%) of the study area. Canyons and mountain slopes (48.5% of the study area) have similar soil and vegetation to the plains, but contain more contours and rocky outcroppings. Isolated meadows (2.6% of study area, ranging in size from a few square meters to 120 ha) are interspersed throughout the landscape wherever water meets the soil surface, and are densely vegetated with saturated soils. Each habitat presents different levels of predation risk for vicuñas. In canyons and meadows, physical structures (rocky terrain, tall vegetation) provide cover for pumas and impede vicuña visibility. Thus, vicuñas spend more time being vigilant in canyons and meadows than in plains (Donadio & Buskirk, 2016) and avoid them at times of the day when pumas are most active (Smith et al., 2019b). Exclosure experiments revealed that the spatial patterning of predation risk results in heavy herbivory and suppression of vegetation in plains but not in canyons and meadows, where vigilance is high and, in canyons, rates of herbivory are low (despite similar forage plant composition and higher palatability compared to plains) (Donadio & Buskirk, 2016). In meadows, water and nutrient availability (bottom-up forces) allow for rapid grass regrowth, dampening top-down effects of grazing. Therefore, differences in responses to a novel top-down force are likely to be indicative of an altered trophic cascade mechanism in plains and canyons.

Description of mange outbreak

Mange is spreading among wild vicuña populations throughout the Andes (Acebes et al., 2018). The cause of its spread remains unclear, but it may stem from contact with domestic animals infected with the disease (Ferreyra et al., 2022). We assessed how the disease progressed throughout the population in SGNP by systematically sampling evidence of mange in vicuñas killed by radio-collared pumas from 2014–2017. Sampling used vehicular surveys to estimate changes in vicuña density from 2004–2020, where signs of disease along transects were noted from 2014–2020 (see Supplementary Material). Herein, we use 'before the mange outbreak' and 'after the mange outbreak' to refer to the periods before and after August 2015 (justification in Supplementary Material), when the disease became prevalent. There is no evidence that the disease has run its course in SGNP. Thus, no fixed period yet represents a time 'after' the disease.

Animal capture and monitoring

We conducted puma captures from April 2014–January 2016 and vicuña captures in April - June of 2014 and 2015 (Permit #DCM 455 and subsequent renewals issued by the Argentine Park Service). We deployed GPS collars on 24 female vicuñas (GPS 6000SD, Lotek) and four female and five male pumas (Iridium Track M2D, Lotek). All puma and vicuña GPS collars had a 3-hr fix rate. To avoid replication in habitat selection analyses, only one adult female vicuña was collared per social group. [See Smith et al. (2019b) for details on puma and vicuña capture methods.] We monitored the fate of pumas and vicuñas from April 2014–February 2017. Kill sites of pumas were field investigated to assess the distribution of vicuña kills and model kill rates over time (see Supplementary Material).

We trapped Andean condors in SGNP with baited cannon net traps and tagged 12 adult birds (Permit #DCM 470) with 70 or 50 g solar Argos/GPS PTT tags (Microwave Telemetry Inc.) from December 2014–February 2017. We programmed GPS PTT units to collect data daily every 60 minutes from dawn until dusk. For more details on condor capture methods, see Perrig et al. (2016, 2020).

Habitat utilisation and kill rates

We resolved whether predation or mange was the ultimate direct cause of the vicuña population crash by examining predator and prey habitat use and kill rates.

Mange could directly kill vicuñas, but may also indirectly increase mortality by rendering vicuñas more vulnerable to predation by limiting mobility. Hence, if pumas continued to be the dominant driver of vicuña behaviour and mortality after the mange outbreak, vicuñas should increase their use of plains to minimise their vulnerability, and pumas should pursue them in plains, increasing kill rates overall. To this end, we calculated the proportions of vicuña locations and kill site locations in plains and the number of kills per month by individual puma for the duration of our GPS data collection.

We evaluated whether the vicuña population decline impacted condor activity by examining changes in space use of individual condors fitted with GPS tags in SGNP. Although increased vicuña mortality could initially benefit scavenging condors, we expected that the eventual vicuña decline would reduce condor use of the park as predation ceased to provide regular food subsidies and vicuña mortalities from mange waned. We calculated the monthly proportions of all condor locations and of individual condor foraging locations inside the park, considering only birds tracked for >5 months (n = 11). Foraging locations were identified by subsetting locations at midday (5–8 h after sunrise) where condor velocity was 0 (indicating that condors were likely feeding on the ground rather than flying overhead; Perrig et al., 2020).

We fit generalised linear mixed-effects models to test whether the proportion of vicuña locations in plains, proportion of kill site locations in plains, total number of kills per month, proportion of total condor locations, and proportion of condor foraging locations in SGNP varied as a function of time since the mange outbreak (modelling details in Supplementary Material). Models in which proportion data were the response variable were fit with a beta-binomial distribution using the *glmmTMB* package (Brooks et al., 2017). The model for the number of puma kills was fit with a negative binomial distribution. Time since mange was demarcated by assigning all months pre-mange outbreak (the baseline) with a value of '0' and all months after mange outbreak with increasing integer values (Table S2) (Brooks et al., 2017); we also tested alternative models that compared the slopes of trends before and after mange outbreak.

Field vegetation sampling and analysis

We predicted that the reduction in vicuña density following the mange outbreak would release vegetation from herbivory in plains; in contrast, we predicted negligible effects in canyons (where herbivory was already low due to predation risk) and in meadows (where high water and nutrient availability maintained high vegetation biomass despite herbivory). We compared prior measurements of standing green biomass, percent cover of graminoids, and height of graminoids within vegetation plots from February 2011 (at the peak of the growing season) to measures in the same plots in February 2019 to test whether vegetation in each habitat responded to the vicuña population crash (see Supplementary Material).

We tested for changes in vegetation following the outbreak of mange by running a series of generalised linear mixed-effects models using the R packages *lme4* and *glmmTMB* (Bates et al., 2015; Brooks et al., 2017). All analyses included year (2011 vs. 2019) as a fixed effect and plot as a random intercept (with the exception of our analysis of the proportion of grasses with seed spikes, for which the response variable was at the whole-plot level). All analyses contained an error structure that accounted for repeated measures (details in Supplementary Material).

Remote sensing and time series analysis

We used satellite imagery to examine whether vegetation change across the study area could be attributed to abiotic (bottom-up) factors rather than the top-down disease effect. We calculated the soil-adjusted vegetation index (SAVI; Huete, 1988) from MODIS Terra 16-day images with a 250 m resolution (Didan, 2015, ORNL DAAC, 2020; details in Supplementary Material). We compared the trend of SAVI five years before and after the outbreak of mange by calculating the SAVI slope of each pixel from August 2010–July 2015 and August 2015–July 2020 using the *raster.kendall* function from *spatialEco* package in R (Evans, 2020). To compare landscape-wide changes in SAVI trends, we averaged all slope values in each time period within the study area. We also compared mean SAVI values within each habitat type in the same 5-year before- and after-mange periods.

We expected that if bottom-up forces were the predominant driver of vegetation change, then changes in plant biomass would track fluctuations in precipitation and temperature more closely than time since the mange outbreak. Therefore, we used historical temperature and precipitation data and remotely sensed imagery to test the relative influence of mange and fluctuations in abiotic conditions on continuous change in SAVI in each habitat. For these time series analyses, we extracted the mean monthly SAVI value for each habitat within the study area and compiled data from March 2000–December 2020 on mean monthly temperature and total monthly precipitation (Chen et al., 2008). We fit time series to the SAVI, temperature, and precipitation data and decomposed the time series to isolate trends independent of seasonality and random variation (Figures S6, S7). We then regressed the SAVI trend values against time since mange outbreak, temperature trend values, and precipitation trend values by fitting a linear GLM. All three covariates in the model were scaled and centered to allow for direct comparisons of coefficients.

RESULTS

Vicuña monitoring and puma kills

Estimated vicuña abundance doubled from 2004 to 2013 [from 8.8 (95% CI 7.4–10.6) to 17.6 (95% CI 14.0–22.1) individuals/km^2; Figure 1a]. Conversely, the vicuña population exhibited a sharp decline after 2017, when estimated density decreased by 90% from 13.7 (95% CI 9.7–19.4) to only 1.3 (95% CI 0.4–4.3) individuals/km^2 in 2020 (Figure 1a). Time since the mange outbreak was positively associated with both the proportion of vicuña locations and the proportion of puma kill sites in plains (Figure S2, Table S2), yet 95% confidence intervals for the slope of change over time (separate models were fit before and after mange) overlapped. However, despite limited evidence that vicuña use and puma predation increased in plains, total monthly kill rates did not change following the mange outbreak, suggesting that the population decline was likely a result of direct, rather than indirect, effects of mange on mortality (Figure 1c, Table S2).

Condor activity

The proportion of total and foraging-specific condor locations decreased over time since the mange outbreak in the best fit models (Figures 2, S3, Table S2). The annual proportion of marked condor locations within SGNP significantly decreased after the outbreak (from 85% in 2015 to 4% in 2018; Figure 2, Table S2), though we note that not all individuals were instrumented throughout the study period. Together, these results suggest that there appears to be a lag effect of the mange outbreak on condor use of SGNP, with condor activity decreasing dramatically in SGNP with time since mange outbreak (Figure 2).

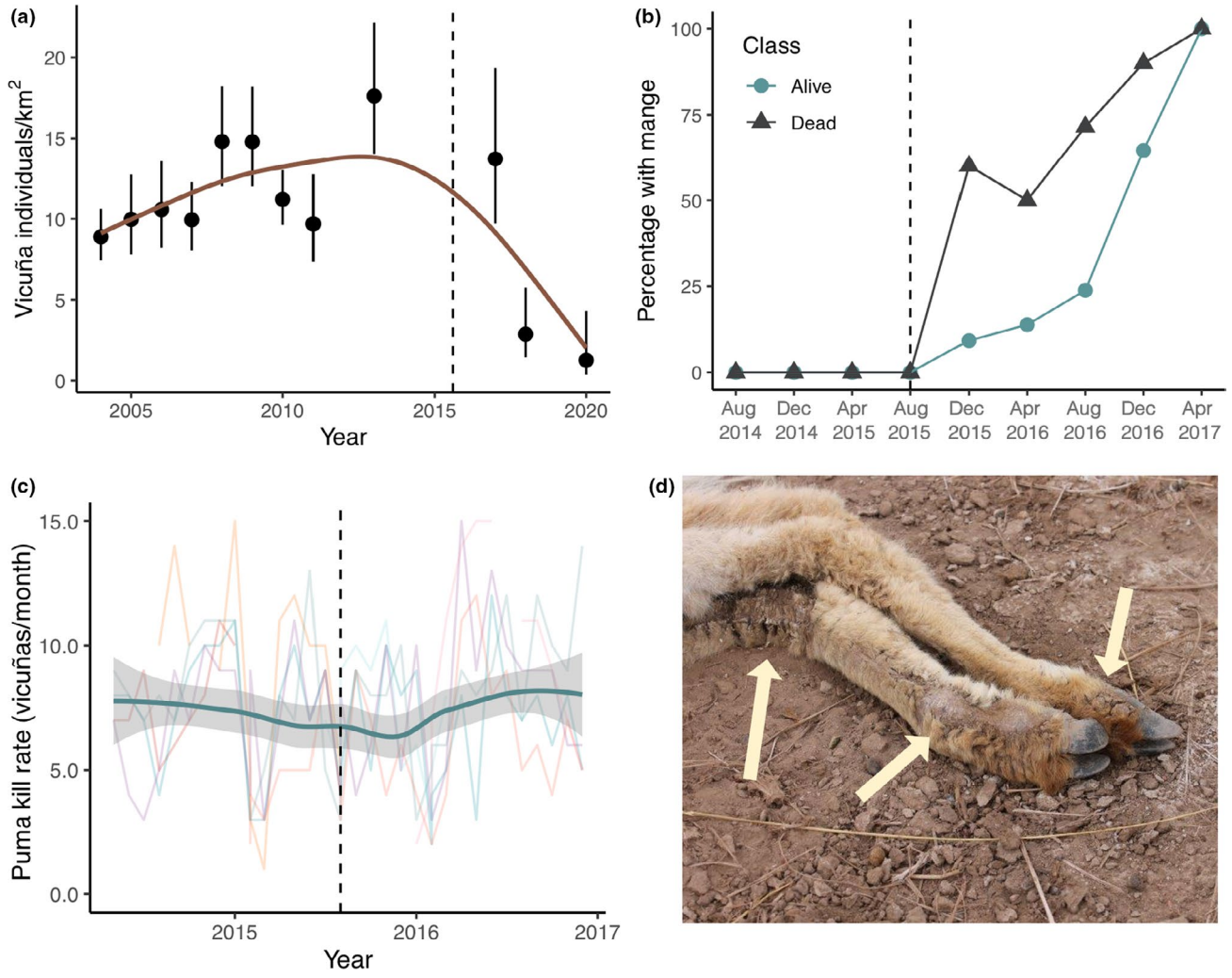

FIGURE 1 Trends in (a) annual mean vicuña densities [95% CI] from distance sampling transects [2017 data obtained from Ferreyra et al., 2022]; (b) percentage of mange incidence in collared vicuñas, in relation to the total number of live collared vicuñas (solid circles) and dead collared vicuñas (solid triangles); and (c) puma kill rates over time in San Guillermo National Park, where thin lines show individual puma monthly kill rates and standard errors of the smoothed pattern are shown in grey. Vertical dashed black lines indicate the outbreak of mange within sampled vicuñas in August 2015. Alopecia from mange on the legs of a vicuña carcass is depicted by yellow arrows in (d).

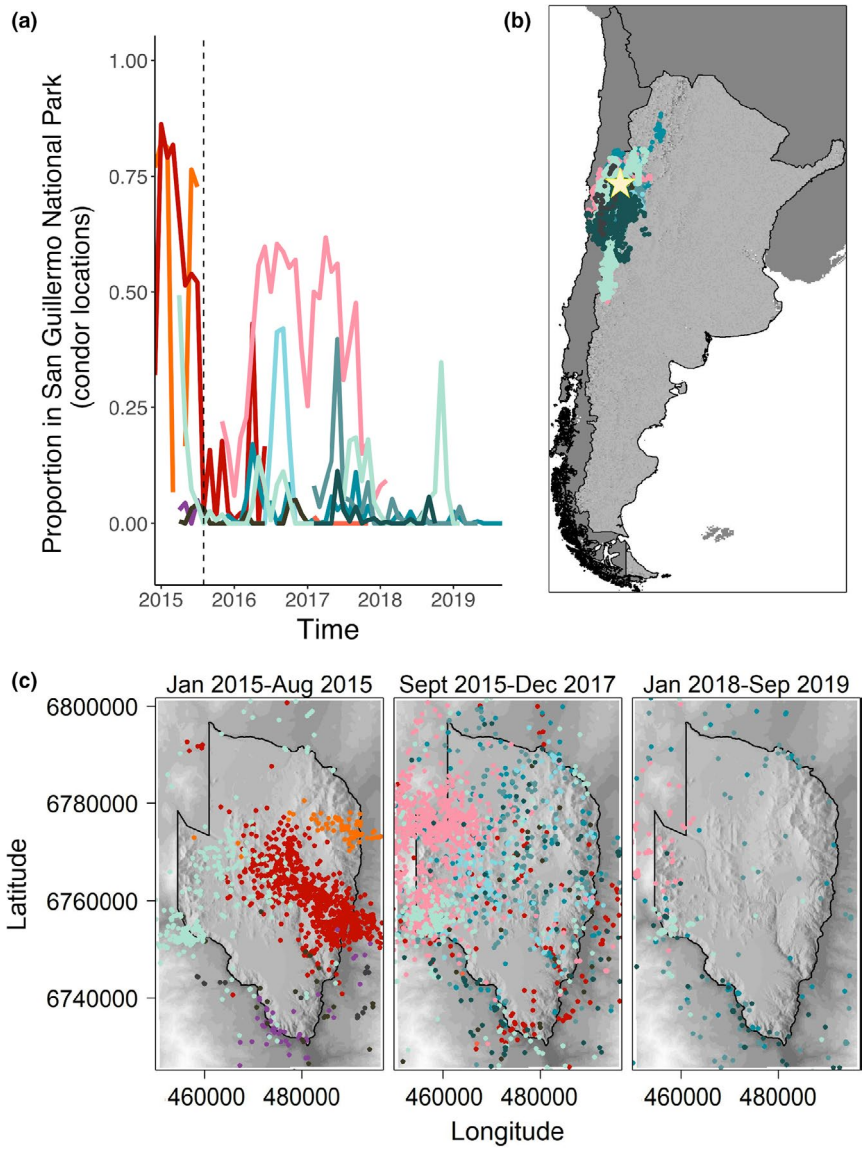

FIGURE 2 Andean condor habitat use in and around San Guillermo National Park (SGNP; January 2015 - September 2019). (a) Proportion of locations from individual GPS-tagged condors (identified by color) significantly decreased inside SGNP after the mange outbreak (dashed line). (b) The full extent of GPS locations from GPS-tracked condors (identified by color) in Argentina (shaded light grey region) and Chile; location of SGNP indicated by the yellow star. (c) Condor locations in the vicinity of San Guillermo National Park (black outlined area) from January - August 2015 (before the outbreak of mange in sampled vicuñas, n=5 tagged condors), September 2015 - December 2017 (after the outbreak of mange, when the vicuña population was in decline, n=10), and January 2018 - September 2019 (after the outbreak of mange, when the vicuña population was reduced to ~1.3 individuals/km^2 and carcasses were rare, n=5).

Field vegetation measurements

Standing green biomass, percent cover, graminoid height, and prevalence of seed spikes all increased significantly in plains after mange, exhibiting 80%–900% increases between 2011 and 2019, but not in canyons and meadows (Figures 3, S4, Table S3). Given that plains constitute 48.9% of the study area, these increases in plant biomass, cover, and height had a dramatic effect on the quantity and distribution of vegetation on the landscape (Figures 3, 4, S4). While grass communities in plains and canyons have similar species compositions, heavy herbivory in the plains, where predation risk is low, maintained stark differences in green biomass, cover, and height between plains and canyons, generating patchiness in plant availability across habitats on the landscape. After the outbreak of mange, vegetation increases in plains largely erased the differences in plant availability between plains and canyons.

Remotely sensed vegetation and time series

After the mange outbreak, the rate of change of green biomass increased 17-fold in the study area (Figure 4). If abiotic conditions were a stronger driver than mange, we

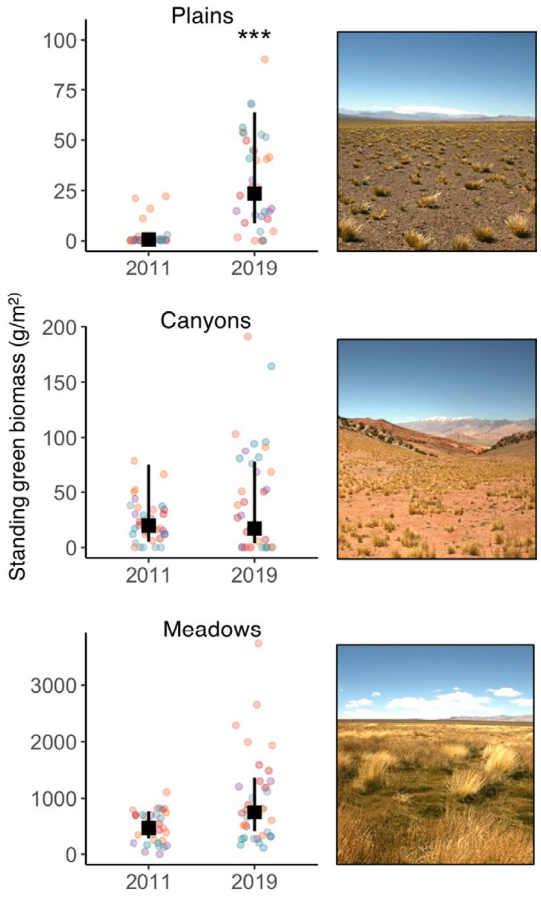

FIGURE 3 Impact of vicuña Sarcoptic mange outbreak on vegetation biomass across habitats in San Guillermo National Park. Standing green biomass (in g/m^2) of graminoids at unfenced control plots (identified by color) is shown for 2011 (before mange outbreak) and 2019 (after mange outbreak) in plains, canyons, and meadows (pictured). Back-transformed predicted means from generalised linear mixed-effects models are shown with 95% confidence intervals (*** denotes $p<0.001$).

would expect some degree of vegetation change across all habitat types, and that in similarly arid canyon and plains habitats these changes would be of a similar magnitude. However, the increase in the slope of the SAVI trend after the outbreak in plains was more than double that in meadows or, notably, canyons (Figure 4b). Moreover, average biomass in plains increased by 7.1% after the outbreak, whereas biomass in canyons increased by 3.0% and in meadows decreased by 0.4% (Figures 4c, S5). For our time series analysis, the covariates examined explained more variation in vegetation in plains than in canyons or meadows (Table S4). Time since the mange outbreak was positively associated with an increase in standing green biomass in all habitat types, but this effect was strongest in plains (Table S4). Furthermore, the best model for green biomass in plains did not include either temperature or precipitation, whereas these variables both remained significant predictors in meadows and canyons models. Finally, when examining effects of time since mange on SAVI alone, time since mange explained 25% more variation in plains than in canyons and over four times more variation than in meadows (Table S4).

DISCUSSION

The mange outbreak in this remote Andean protected area, where baseline data on ecological interactions were available, afforded the opportunity to observe an ecosystem in transition and identify the cascading effects of a pathogen. We show that while both predators and pathogens can trigger trophic cascades, they may initiate different mechanisms of top-down control with altogether divergent effects on whole-ecosystem dynamics (Figure 5). Prior to the mange outbreak, puma predation risk drove vicuña behaviour, largely constraining the effects of herbivory to plains refuge habitat, and provided a steady supply of carcasses to condors (Figure 5). The proliferation of mange throughout the vicuña population disrupted these tight relationships, releasing vegetation in the plains and reducing condor presence and foraging in the park (Figure 5).

We verified that mange was the direct cause of the vicuña population decline by demonstrating that puma kill rates did not change after the mange outbreak. Puma kill rates in SGNP are high (Smith et al., 2020), and it is likely that pumas killed enough vicuñas to exceed their energetic needs prior to the outbreak. While our results suggest that pumas hunted somewhat more in plains after the mange outbreak, this represented a shift in the location rather than the quantity of kills; thus, an increase in predation pressure was not the primary cause of the vicuña decline. We found that the proportion of tagged condor locations inside SGNP decreased substantially after the mange outbreak, following a slight lag. Condors may have been sustained by mange-killed vicuña carcasses in the early stages of the disease outbreak, but fresh carcasses were rare within SGNP after 2017, and condors thus appeared to shift scavenging elsewhere.

Therefore, both in situ measurements and remote sensing indicate that after the mange outbreak, plant biomass (as well as cover and height) increased dramatically in the plains, where herbivory had previously been high, but largely did not change in the high-risk canyons and meadows. This result may have been expected in meadows, which are associated with more productive soils and can recover quickly from herbivory pressure (Donadio & Buskirk, 2016). However, the difference in magnitude of response between plains and canyons, in addition to a lack of evidence for climatic drivers of vegetation shifts, indicate that a disease-induced reduction in herbivory is responsible for the greater release of plants in plains. Thus, the cascading effects of mange undermined the spatial patterns of plant growth created by predation risk and dramatically increased grass biomass and cover in former predation refuges.

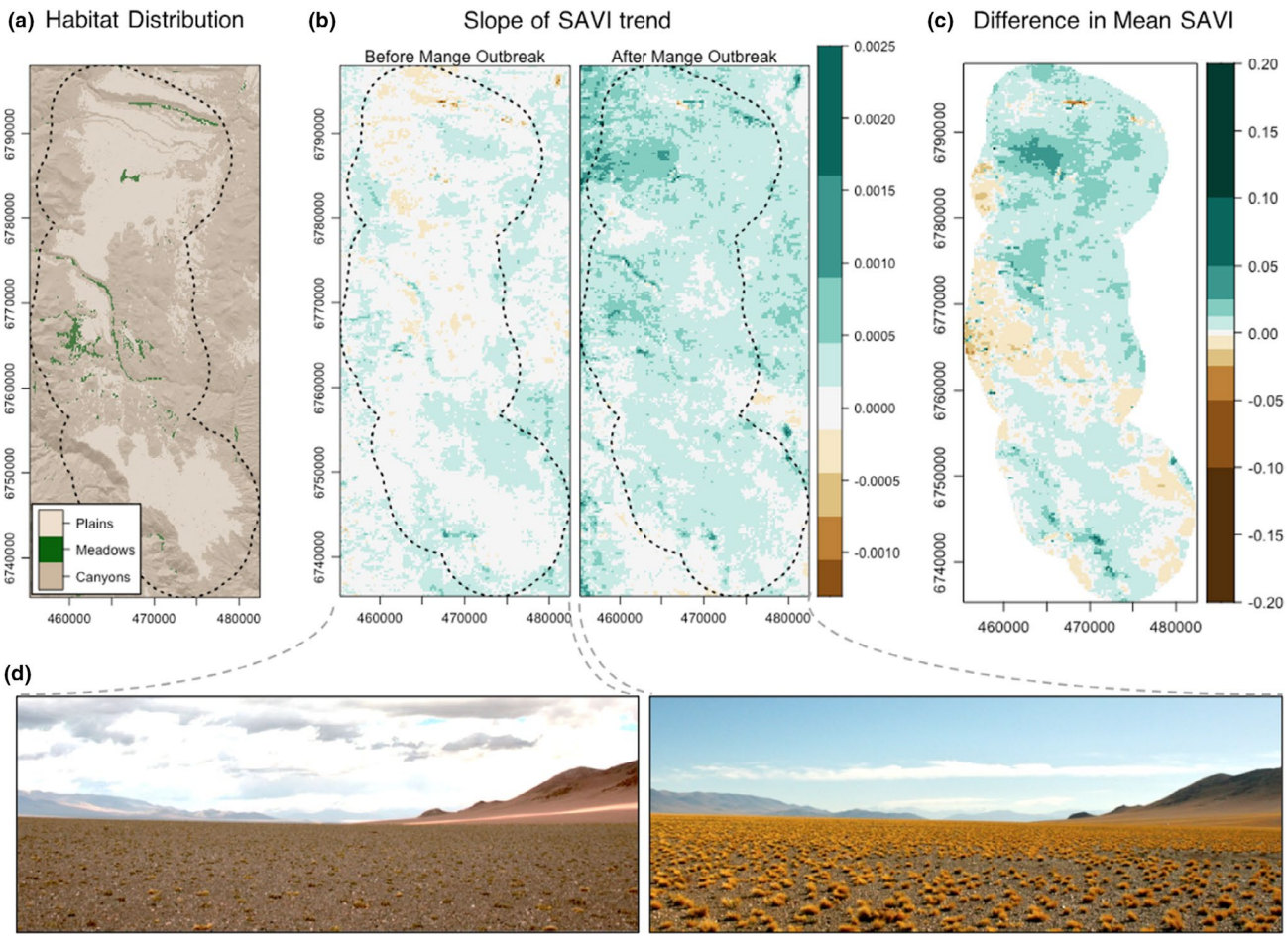

FIGURE 4 Impact of vicuña Sarcoptic mange outbreak on changes in remotely sensed vegetation biomass and distribution in San Guillermo National Park. (a) Map of the distribution of habitat types (plains, canyons, and meadows) and study area (dashed line). (b) Slope of the soil-adjusted vegetation index (SAVI) trend within the time periods of August 2010 - July 2015 ("Before Mange Outbreak") and August 2015 - July 2020 ("After Mange Outbreak"). (c) Difference in mean SAVI between the same five year periods pre- and post- mange outbreak as in (b), represented by a gradient of increases in greenness (teal) to no change in greenness (white) to decreases in greenness (brown). (d) Photos taken during the height of summer in the same spot before and after severe mange outbreak demonstrate stark changes in plains vegetation.

In the long-term wake of this perturbation, the trophic structure of the ecosystem may return to its previous state. However, studies on disease outbreaks in wild animal populations suggest that pathogens could push ecosystems into alternate stable states (Kohler & Wiley, 1997). Hence, ongoing changes in the study system could continue to reveal new ramifications due to the fragility of tightly linked food web interactions (Dobson et al., 2006). The release of vegetation in plains is likely to have further cascading effects throughout the community; increased plant availability could stimulate small mammal and bird population increases, as occurred when large herbivores were experimentally excluded from savanna grasslands (Keesing & Young, 2014). Vegetation increases may also facilitate the recovery of vicuñas by providing widespread forage in a habitat comparatively safe from pumas (Smith et al., 2020). Meanwhile, reductions in condor use of protected areas could have important consequences for human-wildlife coexistence. In nearby agricultural landscapes, condors are susceptible to intentional and unintentional poisoning of carrion (Pauli et al., 2018; Plaza et al., 2019); condors have also increasingly been observed feeding at landfills in the region (Duclos et al., 2020). Pumas could also be at greater risk of conflict with humans and livestock if they, too, move outside the park in response to the reduction of their prey base (Cocimano et al., 2021; Wilkinson et al., 2020); conversely, pumas may instead remain and shift to consuming smaller rodent or lagomorph prey (Osorio et al., 2020). If the latter, apparent competition with nonnative hares could limit the ability of the vicuña population to recover (Barbar & Lambertucci, 2019). Indeed, preliminary data on European hares in the park indicate that they may have increased in density (from ~1–3 individuals/km^2 in 2006–2008 to 5.8 individuals/km^2 in 2020) and in puma diets (from 6% in 2009 to 15% in 2020) in the years after mange (Donadio et al., 2010; E. Donadio, unpublished data); however, sample sizes for these surveys were low, and these patterns should be confirmed with further study. Ultimately, it is likely that the ecological

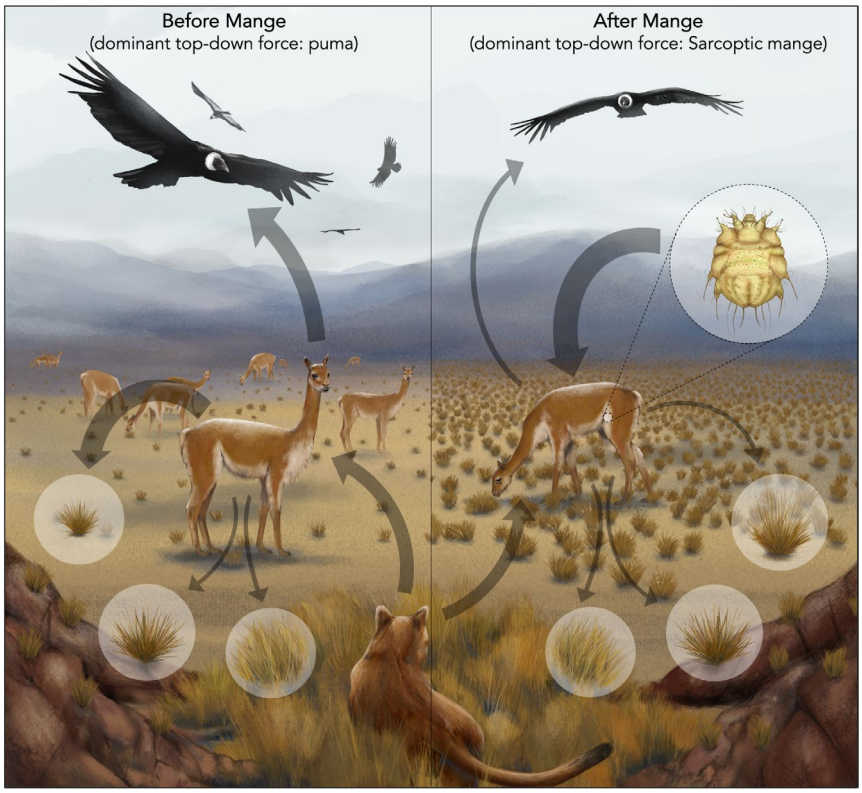

FIGURE 5 Ecological interactions in San Guillermo National Park pre- and post-mange outbreak. Prior to mange outbreak, vicuñas were abundant and avoided canyons due to high predation risk from pumas; vicuñas foraged in meadows during the day when pumas were less active, but high resource availability in meadows maintained high vegetation biomass and cover. Thus, the impacts of herbivory were largely evident in the safer plains, such that plains vegetation biomass, cover, and height were low compared to canyon and meadow vegetation. Andean condors were plentiful in the park as they fed heavily on puma-predated vicuña carcasses. After the mange outbreak, the majority of the vicuña population succumbed to the disease. Thus, vegetation in plains was released from herbivory, homogenising the landscape as plains plant biomass, cover, and height increased while canyon and meadow vegetation did not change. Condors reduced their use of the park as vicuña carcasses became scarce.

effects of the mange outbreak extend well beyond the puma-vicuña-condor-vegetation food web, and the full impacts of the disease on the Andean ecosystem have yet to be fully uncovered.

Despite decades of research on predator-driven trophic cascades, comparatively little is understood about the ecological ramifications of pathogen-driven cascades (Buck & Ripple, 2017). Notably, in this system, predators cause strong behaviourally mediated trophic cascades, suppressing vegetation in refuge habitats where herbivores concentrate. The observed mange outbreak shifted the trophic cascade mechanism to be density mediated, diminishing the spatial patterning of vegetation that had emerged under predator top-down control (Donadio & Buskirk, 2016). Although the contrast in cascading effects of behaviour and density have been demonstrated in experimental mesocosms (Schmitz, 2008; Schmitz et al., 2017), we document a shift from one type of trophic cascade to another due to a transition in the dominant top-down force at a landscape scale. Some work suggests that disease and predators may create different cascading effects; for example, overfishing of apex predators led to increased prevalence of disease in urchins, yet disease did not reduce urchin densities to the same degree that predators did, and thus urchin barrens were maintained even with disease as a top-down force (Lafferty, 2004). However, in this case, both predator- and disease-induced cascades were density mediated. Other compelling work has illustrated the ability for pathogens to cause density mediated trophic cascades (e.g. Lindström et al., 1994; Schultz et al., 2016), yet these effects are not examined in contrast to existing top-down forces. We contribute evidence that disease-induced density mediated trophic cascades result in extremely different outcomes than predator-induced behaviourally mediated trophic cascades, with consequences to other trophic levels.

In almost all cases, disease-induced trophic cascades are density mediated (Buck & Ripple, 2017). When they are behaviourally or trait mediated, the effect is often a result of direct physiological changes that lead to reduced mobility or feeding efficiency (e.g. Haddaway et al., 2012; but see Weinstein et al., 2018 for a discussion of "landscapes of disgust"). In our system, vicuña mobility was impacted by mange infection, which appeared weakly related to habitat utilisation

after mange (Figure S2). However, these effects were swamped out by the rapid near-eradication of vicuñas in the park. Rather, density mediated effects were observed, largely because of their contrast to puma-driven behaviourally mediated effects. Whether stark density-mediated disease impacts will extend to other systems impacted by other pathogens will depend on the transmissibility and severity of the disease; such pronounced trophic cascades are less likely when pathogens spread less quickly or induce lower mortality (e.g. Lafferty, 2004), and under such conditions trait-mediated impacts may be more easily observed. However, mange is becoming increasingly prevalent in many systems worldwide (Escobar et al., 2021), and where it impacts dominant herbivores, similar cascading impacts on vegetation may emerge. Mange also impacts many predators (Escobar et al., 2021), and understanding the community-wide impacts of predator disease, in contrast to herbivore collapse, should be of equal importance to conservation.

Furthermore, we demonstrate that the ramifications of trophic cascade mechanism shifts can manifest in large-bodied vertebrates, a pattern rarely documented at large spatial scales (Peacor et al., 2020). In SGNP, behaviourally and density mediated cascades had strong but divergent effects on vegetation and scavenger activity (Figure 5). Where predation risk was high, the strength of the behaviourally mediated cascade was equivalent to strong herbivore population suppression, as evidenced by the lack of vegetation change in canyons before and after the mange outbreak. In contrast, where predation risk was low, the disease-triggered population collapse dramatically reduced vicuña herbivory (Figures 3 and 4). Importantly, while condor presence was facilitated under predator top-down control, the pathogen-driven cascade led to the functional abandonment of the park by the region's main obligate scavenger (Figure 2). Similar to work on the role of predators in providing consistent food sources to scavengers (Wilmers et al., 2003), our results suggest that the ephemeral pulse of food available to condors immediately post-mange was inadequate to support scavengers in the long term. These knock-on effects likely further extend the spatial impacts of mange to nutrient cycling and associated ecosystem processes, which may be similarly influenced by predator top-down control (Monk & Schmitz, 2021).

CONCLUSIONS

We provide evidence that emergence of a wildlife disease in a dominant herbivore can have cascading effects with profound consequences for the configuration of the ecosystem. The massive shift in plant community dynamics and carbon storage in African savannas following the eradication of rinderpest in wildebeest (Holdo et al., 2009) is one of several prior examples of disease-mediated trophic cascades with such ecosystem-wide consequences (Buck & Ripple, 2017). Here, we show that the magnitude of such a cascade is a direct result of the unravelling of an existing strong predator-prey behavioural interaction and its established ecological outcomes. Furthermore, this outbreak demonstrates the qualitative differences between a predator-driven cascade, which maintained strong habitat-specific, interdependent trophic interactions, and a disease-mediated cascade, whose indirect effects disrupted the food web across the landscape. We argue that examining differential trophic cascade mechanisms across top-down forces is essential to track rapid human-induced ecological change.

Disease transmission between wildlife and domestic animals can be catastrophic for both players, threatening local livelihoods and disrupting natural processes in protected areas (Miller et al., 2013; Rajeev et al., 2017). As land use modification and fragmentation continue to increase interactions between domestic and wild animals, disease-dominated trophic cascades may come to supplant predators as top-down forces in some systems. Our research shows that such shifts in top-down control can not only have marked consequences for infected prey populations, but can also transform the trophic relationships and spatial configuration of ecosystems. Thus, the spread of disease as a novel agent of top-down control can potentially jeopardise the future of ecological processes and species even in protected and remote areas. Developing strategies for human-wildlife coexistence and protected areas management that minimise the exchange and spread of disease will be critical for the conservation of ecosystems sustained by tightly connected food webs as well as the preservation of human health and livelihoods.

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CONFLICT OF INTEREST

Authors declare no competing interests.

AUTHOR CONTRIBUTION

J.A.S., J.D.M. and E.D. conceived and designed the study; J.D.M., J.A.S., E.D., P.L.P., M.F., S.A.L., J.N.P. and A.D.M. collected data; J.D.M., J.A.S., P.L.P., R.D.C., O.R.B. and S.A.L. analysed data; J.D.M., J.A.S., O.J.S. and A.D.M. wrote the manuscript with input from all authors and all authors edited the manuscript.

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