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Insight

Pathogens, disease, and the social-ecological resilience of protected areas

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ABSTRACT. It is extremely important for biodiversity conservation that protected areas are resilient to a range of potential future perturbations. One of the least studied influences on protected area resilience is that of disease. We argue that wildlife disease (1) is a social-ecological problem that must be approached from an interdisciplinary perspective; (2) has the potential to lead to changes in the identity of protected areas, possibly transforming them; and (3) interacts with conservation both directly (via impacts on wild animals, livestock, and people) and indirectly (via the public, conservation management, and veterinary responses). We use southern African protected areas as a case study to test a framework for exploring the connections between conservation, endemic disease, and social-ecological resilience. We first define a set of criteria for the social-ecological identity of protected areas. We then use these criteria to explore the potential impacts of selected diseases (foot-and-mouth disease, anthrax, malaria, rabies, rift valley fever, trypanosomiasis, and canine distemper) on protected area resilience. Although endemic diseases may have a number of direct impacts on both wild animals and domestic animals and people, the indirect pathways by which diseases influence social-ecological resilience also emerge as potentially important. The majority of endemic pathogens found in protected areas do not kill large numbers of wild animals or infect many people, and may even play valuable ecological roles; but occasional disease outbreaks and mortalities can have a large impact on public perceptions and disease management, potentially making protected areas unviable in one or more of their stated aims. Neighboring landowners also have a significant impact on park management decisions. The indirect effects triggered by disease in the human social and economic components of protected areas and surrounding landscapes may ultimately have a greater influence on protected area resilience than the direct ecological perturbations caused by disease.

Key Words: *disease; identity; pathogens; protected areas; resilience; social-ecological systems; southern Africa*

INTRODUCTION

As human influences extend across the globe, natural habitats and the species, communities, and ecosystems that they contain are increasingly under threat. Land cover change and habitat fragmentation are consistently listed among the most significant threats to biodiversity, and as key drivers of the currently high extinction rate of species (Pimm et al. 2014). The international conservation community has responded to the ongoing loss and degradation of natural habitats in a variety of ways, but protected areas remain central to current strategies (Chape et al. 2005). Among the 20 Aichi Biodiversity Targets of the Convention on Biological Diversity, Target 11, for example, states that "by 2020, at least 17 per cent of terrestrial and inland water, and 10 per cent of coastal and marine areas, especially areas of particular importance for biodiversity and ecosystem services, are conserved through effectively and equitably managed, ecologically representative and well-connected systems of protected areas and other effective area-based conservation measures, and integrated into the wider landscapes and seascapes" (CBD Secreteriat 2010:9). As this definition highlights, protected areas are not only central to strategies for halting biodiversity loss through their roles in recolonization, restoration, and ecological landscape connectivity (Bengtsson et al. 2003, Chape et al. 2005) but also play an important role in contributing to human well-being and other developmental goals through the provision of ecosystem services.

Given the relevance of protected areas to global development and sustainability goals, it is important to recognize that protected areas may themselves be vulnerable to certain kinds of social, ecological, and economic change. Threats to protected areas and the biodiversity that they are intended to conserve can come from within the protected area (e.g., overstocking of game species), from the surrounding landscape (e.g., invasive species, poaching, changes in surrounding land use, and loss of connectivity between populations resulting in loss of genetic diversity and population viability; Woodroffe and Ginsberg 1998, Laurance et al. 2012, Laurance 2013), and from ecological, social, and economic systems at multiple scales (e.g., loss of ecologically important disturbance regimes, poor governance, international conflict; Ervin 2003, Laurance et al. 2012). In turn, degradation of protected ecosystems may have consequences for human health and well-being that extend far beyond their boundaries (Foley et al. 2005). Protected areas are thus fundamentally social-ecological in nature (Palomo et al. 2014), with ecosystem dynamics both driving and being driven by management and institutional agendas at various different levels and scales (Cumming et al. 2015*a*).

Consideration of protected area vulnerability brings us into the domain of social-ecological systems theory. One of the more widely adopted organizing concepts in this context is that of resilience. Protected area resilience, in the sense of specified, normatively positive resilience, can be thought of as the ability of the social-ecological system to maintain key elements of its identity in space and time, through various perturbations and changes (Cumming et al. 2005). It is important to note that resilience, as used here, does not imply that systems should bounce

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back to desired states after perturbations, but rather that they will persist with change in desirable states, manifesting as maintained identity. We consider "protected area identity" to be linked explicitly to their stated aims, as decided upon by society. In a conservation context, "protected area identity" particularly refers to the suite of species and habitats that the area is expected to conserve; but also, potentially, the provision of a range of supporting, regulating, provisioning, and cultural ecosystem services (Cumming et al. 2005, 2015*a*).

Although some elements of protected area resilience have been extensively researched, e.g., economic effects on surrounding land use, and the value of surrounding ecological buffer zones (see Payés et al. 2013, Palomo et al. 2013, Strickland-Munro and Moore 2014), others are less frequently considered. One such element of protected area resilience in social-ecological research is that of disease. Although veterinary concerns may have major impacts on the management of protected areas, disease issues are often considered distinct from conservation problems (Deem et al. 2008). This may be because veterinarians and ecologists work for different management agencies, each with their own scientific culture and approach to research (Deem et al. 2008).

Although there are comparatively few articles on resilience and disease published in mainline conservation, ecosystem services, and resilience journals, the relatively young fields of landscape epidemiology (Meentemeyer et al. 2012, Myers et al. 2013) and ecohealth (e.g., Berbés-Blázquez et al. 2014) have produced a swath of recent literature contextualizing the social-ecological and complex nature of disease. The one health paradigm (D'Amico Hales et al. 2004, Cumming and Atkinson 2012, Cumming and Cumming 2015) in particular promises to be influential in the development of new conservation policies and strategies that recognize that human and ecological health are inherently linked. By more explicitly linking veterinary and human medicine and including biodiversity conservation perspectives in the control and management of disease (Olival et al. 2013, Cumming and Cumming 2015), the one health literature has illuminated the complex links between land degradation, disease management, and human health and the relevance of multiple institutional levels and spatial scales for disease-related land management (Meentemeyer et al. 2012, Myers et al. 2013). As Berbés-Blázquez et al. (2014) point out, one health and resilience thinking are both rooted in complexity science and regard human and nature as coupled, social-ecological systems. Combining insights from these two schools, as well as landscape epidemiology (Meentemeyer et al. 2012), has the potential to meaningfully improve our understanding of human and ecological health in the face of changing environmental conditions (Berbés-Blázquez et al. 2014).

In this article we place the ecohealth proposal, that disease management is a social-ecological concern (Olival et al. 2013), into the resilience framework. We do this by considering the relevance of pathogens and the diseases that they cause, for the resilience of protected areas, using southern Africa as an illustrative example. Our primary goal is to provide a better integration of disease into the growing literature on the resilience of protected areas. We focus our argument around the premises that wildlife disease (1) is a social-ecological problem, and hence is best approached from an interdisciplinary perspective; (2) has the potential to lead to changes in the identity of protected areas; and (3) interacts with conservation both directly, via impacts on wild animals, livestock, and people, and indirectly, via the public, conservation management, and veterinary responses.

Disease and social-ecological identity of protected areas

Cumming et al. (2005) and Cumming (2011*a*) have argued that the resilience of a social-ecological system can be understood as its ability to maintain its identity in both space and time. This definition has some operational advantages over alternative uses of "resilience," e.g., as the return time of a system to equilibrium following a perturbation or the ability of a system to retain the same structure and controls (Holling 1973) in situations where the availability of detailed time series information is low, and/or a large number of potential variables may influence resilience (Cumming 2011*a*). Cumming et al. (2005) considered identity in terms of four primary criteria: (1) the components composing the system; (2) the relationships between these components; (3) the sources of innovation that help components and relationships persist via adaptation; and (4) the sources of continuity, such as memory, that contribute to the persistence of the system through time (Fig. 1, Table 1). Ideally, given that it is extremely difficult to think about or build resilience in relation to all possible future events, identity elements should also be considered in relation to a specified set of perturbations.

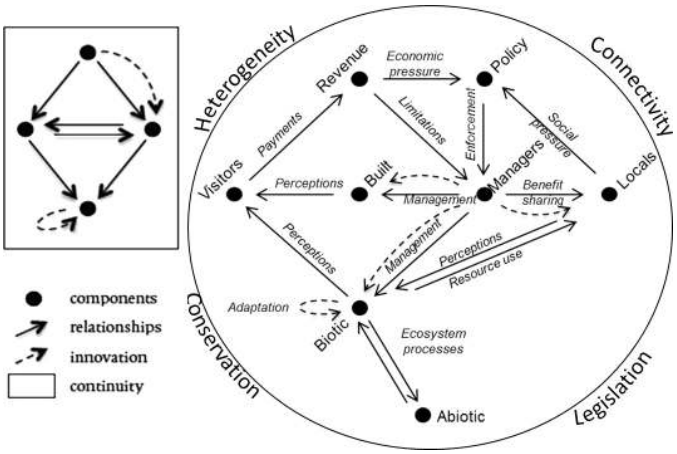

Fig. 1. Conceptual diagram of identity elements of protected areas in healthy systems. Perturbations such as wildlife disease may affect one or more elements directly, and may subsequently ripple through the system via the various connections.

Acknowledging that pathogens are also a part of an ecosystem's identity (Castello et al. 1995, Ayres and Lombardero 2000), the first element of protected area identity, the system components, encompasses the settings and actors present in, or interacting with, the protected area (Table 1). These include ecological components such as the biotic environment, e.g., species composition and abundance, and the abiotic environment, e.g., soil type, nutrient loading, water availability, and local climate, that supports the biotic environment. Social components are also inherent within protected area identity, and include the manager who makes decisions on park operations; protected area users, e.g., tourists and hunters, who visit and recreate in the park; as well as local communities on the periphery of the protected area, who are in some way connected to

Table 1. The primary elements of protected area identity as defined and described in the text.

Element	Attribute	Details
Components		
(objects, agents, entities that make up the system)		
	Biota	Species; biodiversity; community composition and abundance; endogenous parasites and pathogens
	Abiotic environment	Soil structure, nutrients, water, climate
	Built environment	Fences, roads, day and overnight visitor facilities, including infrastructure like dip-tanks and fences built for disease control
	Manager(s)	Person(s) who manages the protected area
	Area visitors	Tourists, hunters, researchers, educators
	Local communities	People that live on the protected area's periphery
	Institutions	Relevant policy, legislation, including policies dealing with disease management.
	Revenue	Income generated by the protected area
Relationships		
(process or interaction variables that link components)		
	Ecosystem processes	Predator-prey relationships, inter- and intra-species competition, the interaction between the biotic and abiotic environment, nutrient cycling, host-parasite dynamics.
	Management	Actions taken by managers regarding the built and biotic environment, as well as local communities in the form of benefit sharing activities.
	User perceptions	A perception about the protected area's biotic and built environments from people who use it, including perceptions about diseases, potentially disease-carrying vectors, and disease-management infrastructure.
	Local community perceptions	Perceptions about the protected area from people who live around it.
	Payments	Money paid by users to the protected area
	Political pressures	Pressures exerted by people (such as local communities) on authorities, which result in policy/ legislation changes.
	Economic pressures	Pressures exerted on ecosystems, via managers, as a result of economic goals (e.g., not making a profit/desire for greater profit/not meeting targets).
	Enforcement	The process by which legislation compliance is ensured
Innovation		
(variables that relate to the development of novel solutions and responses to change)		
	Biological adaptation	Past speciation events; mutation and selection; shifts in ecological community composition that occur in response to environmental changes; acquired immunity
	Social adaptation	Novel policies, learning, information sharing, medical advances, and technology
Continuity		
(variables that maintain identity through time)		
	Legislation	Local, national, international environmental instruments; gazettement status.
	Conservation targets/objectives	Long-term goals that provide continuity in management strategies
	Social capital and memory	Age of protected area and resulting path dependence in terms of visitor expectations, loyalty, and community support
(Spatial) Continuity		
Spatial context and landscape position		
	Where the protected area is, how large, what connected to?	Protected area size, connections between ecological patches; connections between managers and between users; institutional context (Cumming et al. 2015*a*)

or affected by the park. Other components are the infrastructure present in protected areas, e.g., fencing, roads, and accommodation, which is important for supporting the social and ecological components; and the policy arena in which protected areas operate, including environmental, tax, and tenure policy directly relevant to protected areas, as well as policy indirectly relevant through, for example, its implications for visitor rates to a country or region. Finally, revenue is a key component of protected area identity, defining its financial opportunities and limitations.

Social, ecological, and social-ecological relationships between system components, the second elements of identity, maintain, alter, and/or connect the system components. They are the links through which a potential disturbance to a social-ecological system can cascade. Ecosystem processes, e.g., primary productivity, trophic interactions, inter- and intra-species competition, facilitation, decomposition, and nutrient cycling, structure communities and therefore play a key role in shaping the biotic and abiotic environment. These processes include relationships between different pathogen species and their hosts. Management alters or maintains the biotic, abiotic, and built environments, which in turn can influence user perceptions and thereby determine payments made by users to visit the protected area. Economic pressures arising from protected area revenue can determine future management actions and feed back into policy. Policy enforcement and local community perceptions of a

protected area, e.g., problem animals, resource availability, presence of, and susceptibility to, disease, influence protected area management. These perceptions may drive communities to exert societal pressures that alter policy, or partake in (il)legal resource use within park boundaries. Both of these actions either directly or indirectly affect the biotic environment. Finally, managers can mediate community perceptions via benefit sharing schemes and/or via comanagement of the biotic environment.

The third and fourth elements of system identity refer to those adaptation/innovation and continuity processes that maintain system components and relationships. Ecological adaptation is reflected by past speciation events and current genetic, species, and functional diversity. Biodiversity, which includes disease-causing organisms, thus determines the potential for ecological adaptation at the community level, and is an important source of innovation within the biotic environment. Innovation in the social identity of a protected area can arise through novel policies, learning, information sharing, medical advances, and technology; all of which introduce adaptation into management decisions. Heterogeneity in species distributions, genotypes, and ecological functions, as well as connectivity between different parts of a landscape, are sources of continuity. Sources of continuity that promote the persistence of the social identity of a protected area exist in the form of legislation, management goals, and conservation targets, as well as connectivity within management networks.

Disease as a driver of change

Based on our working definition of protected area identity, Figure 1 depicts a hypothetical "typical" protected area system, showing its components, the relationships between them, and elements of continuity and innovation. Pathogens can be viewed as system components that affect other system components and the relationships between them, alter spatial and temporal continuity, and facilitate innovation and adaptation.

In regard to maintaining regulatory processes, pathogens do not only affect elements of system identity negatively. They may not always manifest as disease (Ayres and Lombardero 2000, Tompkins et al. 2011), and may play important roles in regulating ecosystem function (Anderson and May 1978, Hudson et al. 2006) and shaping ecological diversity (Van der Putten and Peters 1997, Holdo et al. 2009, Tompkins et al. 2011). For example, fungal pathogens contribute to tropical forest diversity by disadvantaging species where they are locally common (Bagchi et al. 2014) and managers use disease-causing agents to control invasive alien species (Palmer et al. 2010, Post et al. 2010). Pathogens may also play an important part in maintaining continuity: many of Africa's savanna landscapes owe their ecological integrity to the presence of tsetse flies (*Glossina* spp.) that made these areas initially unattractive to settlement (Rogers and Randolph 1988, De Klerk et al. 2004). Pathogens also promote elements of innovation: infected individuals may develop immunity against disease (e.g., Pienaar 1961), and research and learning may facilitate medical advances and novel policies (e.g., Cumming et al. 2015*b*).

Pathogens also have many negative impacts on protected area components, the relationships between components, and on aspects of continuity and innovation. The most obvious way in which pathogens can affect protected area resilience is by directly impacting biotic components and neighboring human, plant, and animal communities. Pathogens can cause high mortality (Osofsky et al. 2005), morbidity, and suppressed reproduction (Ryan and Walsh 2011), reducing population densities of endangered or keystone species (Gascoyne et al. 1993). Pathogens may also affect economic and social components of protected areas, via their relationships with affected biotic components. For example, disease may lead to reduced tourism visitation to protected areas via altered visitor perceptions (Rittichainuwat and Chakraborty 2009) or, where livestock is affected, negatively impact economic earnings from agricultural production in the surrounding matrix (Pendell et al. 2007). This can, in turn, facilitate the development of unbalanced economic policies and costly management interventions (Coker et al. 2011, Thomson et al. 2013), and thereby influence elements of continuity.

The development of measures to curtail the spread of a pathogen may manifest in national legislation, resulting in new policies (an element of temporal continuity) that are enforced into the future, often at the expense of relationships between managers and local communities. For example, foot and mouth disease (FMD) outbreaks in southern Africa have resulted in the development of policies that dictate that game-proof fences should be erected to control the movement of wild and domestic cloven-hoofed animals (Scoones et al. 2008, Cumming et al. 2015*b*). These fences act as barriers to many ecological processes and FMD outbreaks have therefore also indirectly affected the spatial continuity of many protected areas in southern Africa, and have significantly impacted relationships between conservation authorities, farmers, tourists, and local communities (Cumming et al. 2015*b*). As with many elements of pathogen impacts, however, the story is complex: game-proof fences also reduce human-wildlife conflict and may be a key factor in the success of the conservation of large predators, particularly lion, in southern Africa (Packer et al. 2013). Pathogens may also affect continuity in other ways. Conservation managers may consider pathogens a threat to species conservation, and consequently incorporate preventative measures in long-term goals and management strategies, sometimes with unintended consequences. For example, Chauvenet et al. (2012) modeled the long-termed effects of vaccination of the Serengeti ecosystem's lion population against canine distemper virus (CDV), showing that vaccination may lead to the extinction of the local cheetah population within 60 years.

Elements of relationships between components (e.g., ecosystem processes, user perceptions, economic pressures), innovation (e.g., species adaptation, novel policies, veterinary advances), and continuity (e.g., ecological connectivity, legislation) play a role not only in how pathogens alter components of protected areas, but may themselves be changed by a pathogen outbreak. For example, the biotic environment of a protected area is fundamental to the functioning of a number of its social, ecological, and social-ecological components and relationships (Fig. 1). Changes to the biotic environment may therefore have significant impacts on a number of other attributes of identity, including the perception of users and local communities, revenue payments, political and economic pressure and management.

Direct influences often alter identity elements in an expected way, but system internal relationships and dependencies may create chain reactions or feedback loops with unforeseen impacts. As this

discussion suggests, many of the largest impacts of pathogens on protected area resilience come from indirect effects that are produced as a consequence of the complex and dynamic interactions between components, relationships, innovation, and continuity (Tompkins et al. 2011). This is true even in purely ecological contexts. For example, reductions in predator populations in Yellowstone Park in the United States had negative impacts on surface water quality via a trophic cascade (Ripple and Beschta 2003, Beschta and Ripple 2009). Indirect and cascading effects resulting from pathogens, in both social and ecological systems, can create unexpected consequences for protected area managers and represent a challenging management problem from the perspective of building protected area resilience.

The complexities of understanding the indirect social-ecological impacts of pathogens are further exacerbated by the problems of scale and boundaries. Protected areas exist within a larger biophysical space and social-ecological context, which pathogens can move into and out of. Disease may be contained within the boundary of a protected area, but often will spread from pathogens moving into a protected area system, or move from a protected area to the surrounding matrix. This happened, for example, when bovine tuberculosis spread from south of South Africa's Kruger National Park across the country's border into Gonarezhou National Park in Zimbabwe (Michel et al. 2006, Cross et al. 2009, de Garine-Wichatitsky et al. 2010). Legitimate concerns about the spread of disease between areas and the loss of local disease resistance in wild and domestic animals may be either unnecessarily exaggerated or foolishly ignored by protected area managers, resulting in actions that create additional social-ecological feedbacks and lead to tighter-than-necessary linkages between social and ecological elements of the system. For example, wildlife translocations represent a growing enterprise in South Africa (Goss and Cumming 2013) and the economic gains resulting from selling and stocking wildlife often override considerations about genetic mixing, local adaptation, and parasite and pathogen spread.

The negative effects that pathogens may have on ecological, social, and economic system components are therefore influenced by the existing system configuration, i.e., the relationships and reinforcing feedbacks between components, as well as continuity and potential sources of innovation (Fig. 1, Table 1). The effects of pathogen-driven perturbations on protected area resilience are likely to be further influenced by the way in which a disease outbreak manifests in space and time, and its epidemiological dynamics as determined by the virulence of the pathogen, its incubation period, transmission, epizootic status, existing prevention, and treatment options (Table 2).

Protected area resilience and disease in Southern Africa

Although we have used "pathogen" in a generic sense so far, the impacts of pathogens may depend heavily on their individual properties. To move toward a deeper understanding of the implications of pathogens for protected area resilience, we next consider in greater depth the example of southern Africa and the ways in which different kinds of pathogens can affect southern African protected areas. For this discussion, southern Africa includes the area south of the Zambezi-Kunene axis: South Africa, Zimbabwe, Namibia, Botswana, southern Mozambique, Lesotho, and Swaziland. We focus our analysis on statutory National Parks and Nature Reserves.

Regional Background and History

Southern Africa is a highly biodiverse region and supports an array of indigenous large mammal species (Pimm et al. 2014). Its human population has grown twenty-fold over the last century (UN 2007), with over half of the region's population living in rural areas (UN 2003). Extensive domestic animal production predominates over cultivation in semiarid areas, which compose 60% of the region's 3.4 million km² (Cumming and Atkinson 2012).

Epidemics of contagious bovine pleuropneumonia in the 1850s (Dupuy et al. 2012), rinderpest in 1896-97 (Mariner et al. 2012), and overhunting led to severe depletion of herds of domestic stock, as well as wildlife (Dupuy et al. 2012, Mariner et al. 2012). Protected areas were therefore established for wildlife (Cumming 2004) using fences to separate wild and domestic ungulates (D'Amico Hales et al. 2004). The commercial and subsistence use of wildlife was banned, thereby alienating commercial and communal farmers from wildlife (Dupuy et al. 2012). In many southern African countries, legislative changes in the 1960s gave private land-owners the right to manage and benefit from their wildlife (Bond et al. 2004) and resulted in a diversification of the scale, type, and ownership of protected areas in the region. Protected areas in southern Africa now include private, community, and state-owned initiatives, and range from small-scale (< 15,000 ha) game reserves, to large-scale Transfrontier Conservation Areas, which span multiple countries. Although exact data are hard to come by, nature-based tourism contributes substantially to the region's economy (Scholes and Biggs 2004).

Many livelihoods in rural southern Africa are therefore dependent on two, frequently co-occurring enterprises: wildlife-based tourism in protected areas and domestic animal production (both commercial and subsistence). The resultant interface between domestic animals, people, and wildlife can give rise to human-wildlife conflict (Cumming and Atkinson 2012) including conflict that arises as a result of diseases moving across the interface. The nature of the human-wildlife conflict attributable to a disease will depend on social and ecological factors such as disease epidemiology (Table 2) and protected area or domestic animal management. Within southern Africa, multiple diseases with varying epidemiologies have been recorded within and/or around protected areas (Cumming and Atkinson 2012). This allows us to explore a variety of disease-related effects on protected area resilience.

Relevant pathogens and their implications for protected area resilience

To explore the ways in which disease may influence protected area resilience, we considered how protected area identity (Table 1, Fig. 1) might be affected by each of seven focal diseases common to southern African protected areas. Diseases were selected as case studies based on their perceived relevance to southern African protected areas as well as for a diversity of traits as identified in Table 2. Our chosen case study diseases are FMD, anthrax, malaria, rabies, rift valley fever (RVF), trypanosomiasis (sleeping sickness), and canine distemper (CD). Of these diseases,

Table 2. A typological table of factors of disease epidemiology and management that affect how diseases impact on protected area identity and resilience.

Factor	Description	Implications for impacts on resilience
Pathogen	Viruses, bacteria, fungi, protozoans, arthropods, helminths.	Different taxa have different cellular structures, metabolic pathways, and reproductive mechanisms. Thus, disease control measures, e.g., immunizations, differ for varying pathogen types. The biological make-up and mutation rate of taxa may affect available treatment, and consequently, available management options.
Transmission mode	Contact (both direct and indirect contact as well as horizontal and vertical); Vehicular (via fomite); Vector (either mechanical or biological) and intermediate hosts	Method of transmission affects how easily a disease can spread, which will determine the time and economic resources needed to contain and control it. Feedbacks from these disease control structures can have wide-ranging socioeconomic as well as ecological implication, e.g., fences that impede herbivore migration.
Virulence	The degree of pathogenicity of a parasite in a particular host, and the methods in which it affects it, e.g., by attaching to cells, immunoevasion, immunosuppression, obtaining nutrition from the host, etc.	Virulence factors influence the severity of a disease within its host. More pathogenic diseases may be more likely to kill their hosts, which may impede its transmission into or out of protected area, but may also lead to widespread negative social perceptions.
Disease occurrence characteristics	Enzootic/endemic or epizootic/epidemic	Diseases in enzootic or endemic phases may have less of an acute and/or lethal effect on wildlife populations as well as a decreased perceived socioeconomic impact compared with those in epizootic/epidemic phases.
Host range	Pathogens may be sylvatic pathogens that circulate persistently in some wild species and occasionally spill over to other domestic species, zoonotic diseases that spill over from wildlife to humans, anthropozoonoses (human to animal spread), enzootic (circulate persistently within animal populations). Environmental pathogens refer to those that remain dormant outside of a biological host with environmental factors influencing their survival and emergence as epizootics	Diseases that are zoonotic may have a greater social impact than diseases that are transmitted between wildlife, or between wildlife and domestic animals. Diseases that occur mostly in wildlife, but affect domestic livestock may have important economic impacts. By the same token, diseases that occur in domestic animals or humans may have important ecological impacts.
Incubation period	Diseases may have long or short incubation periods	Diseases with longer incubation periods may take longer to detect and hence may be more difficult to manage.
Wildlife disease control	Culling, sterilization, and vaccination	Different management strategies may have different economic costs, social- and long-term ecological impacts. They may also be more or less effective at disease control depending on how they affect the susceptible population size.

rabies and CD have been introduced into the region. We provide typological context (based on Table 2) for each disease in Table 3, and explore the effects of each on specific elements of protected area identity in Table 4. A more detailed description of how we arrived at these effects are provided in a case-study table in Appendix 1, as well as in Figure 2, which shows the protected areas considered for each case study disease.

FMD in southern Africa is caused generally by the South African Territories serotype virus *Aphthovirus* spp. that is enzootic in free-living buffalo (*Syncerus caffer*) that become infected as young calves, thereby forming part of the ecological memory of the system. During the period of acute infection the virus is excreted, which can cause infection of other susceptible cloven-hoofed species such as impala (*Aepyceros melampus*), that then spread the disease to other wildlife both within protected areas and, because impala have the ability to jump cattle fences, also to cloven-hooved domestic animals in surrounding areas (Vosloo et al. 2009). Although it normally has little effect on indigenous wildlife, FMD can result in significant production losses in domestic stock (Thomson et al. 2003). When FMD outbreaks occur in designated FMD-free areas in southern Africa, they are sometimes managed through compulsory slaughter of infected

Fig. 2. Map of southern Africa, as defined in this study, highlighting national parks (NP) and nature reserves (NR) used to analyze case study diseases. Major national parks and nature reserves in the region are shown in grey. Created with ArcGIS 10.1.

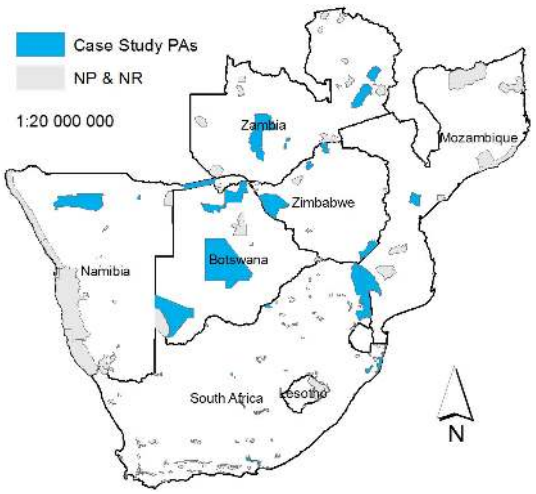

Table 3. Case study diseases considered in this analysis, chosen for their significant impact on the resilience of southern African protected areas in the past 100 years, and to be typologically representative (as outlined in Table 2).

Disease and short description Pathogen	Transmission new	Virulence	Disease occurrence characteristics	Host range	Incubation period	Wildlife disease control
In southern Africa, foot and mouth disease (FMD) is caused by South African Territories serotype virus (SAT, *Aphthovirus* species).						
Virus	Direct contact; Indirect contact (aerosol, fecal-oral), vehicular	Animals: Domestic host - significant production loss and calves can have significant mortality rates. Has less impact on indigenous livestock in southern Africa. Wild host - little if any effect	Epizootic in domestic cattle; enzootic in wildlife (buffalo of importance)	Sylvatic pathogen, enzootic in domestic populations if uncontrolled	2-21 days (animals)	Vaccination, culling, and preventing contact between infected wildlife and susceptible domestic hosts (fences, movement control)
Anthrax is caused by the Gram-positive, rod-shaped, spore-forming bacterium *Bacillus anthracis* that can form dormant endospores for decades.						
Bacteria	Direct and indirect contact (inhaled, ingested, or absorbed) depending on phase of bacterium.	Humans and livestock: Cutaneous anthrax (skin lesions): 5-20% mortality without treatment Pulmonary anthrax (inhalational): Approaching 100% mortality without treatment	Epizootic	Environmental, zoonotic during host phase	1-5 days (humans) 3-7 days up to 14 days (animals)	Burning and burying animal carcasses, antibiotics for humans.
The Malaria parasite *Plasmodium* is transmitted from one human to another by the female *Anopheles* mosquito when it takes a blood meal as a prelude to the reproductive process.						
Parasite	Vector (biological - mosquito)	Humans: Virulence varies by malaria parasite with *Plasmodium falciparum* causing almost all fatalities. Children significantly more vulnerable.	Endemic and requires susceptible human population for maintenance	Enzootic	7-30 days (humans)	Spraying dwellings, reducing areas of stagnant water, or adding film of oil (which kills the larva), using repellents and treated mosquito nets, prophyllaxis. There is not yet any immunization.
Rabies is a viral zoonosis caused by a negative-stranded RNA.						
Virus	Direct contact	Humans and animals: 100% mortality with very few successfully treated cases in humans.	Enzootic with humans as dead end hosts	Sylvatic pathogen	< 1 week to > 1 year	Vaccinating domestic dogs and cats and culling of infected hosts.
Rift Valley Fever is a mosquito borne disease, caused by the Rift Valley Fever RNA virus (RVFV, *Phlebovirus* spp.), which can result in spontaneous abortion or death in ungulates, and flu-like illness or death in humans (NHLS 2011).						
Virus	Direct contact (infected tissue) plays major role in zoonotic transmission; Vector (biological - mosquito)	Livestock: Varies by species but causes almost 100% abortion rate. Sheep are most susceptible with over 90% mortality in young animals and as low as 10% in adult animals. *Humans*: Mortality varies widely during epidemics, but less than 1% total.	Epizootic with long interepizootic periods	Environmental (within eggs of relevant mosquito vector) with potential low level sylvatic cycle, zoonotic during epizootic phase of disease	2-6 days (humans) < 3 days (livestock)	Immunization of livestock
Canine distemper virus (CDV) is a negative-strand, nonsegemented RNA morbillivirus (Gowtage-Sequeira et al. 2009).						
Virus	Direct contact (aerosol, body fluids), vehicular transmission via contaminated food and water	Animal: Varies on immune status of infected animal but thought to vary from 50% to > 90% depending on species.	Enzootic and epizootic phases depending on proportion of susceptible hosts	Sylvatic pathogens, enzootic circulation in domestic dogs	1 week to 1 month (animals)	Block contact between domestic dogs and wildlife, vaccination of domestic dogs

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Human African trypanosomiasis (HAT), also known as sleeping sickness is a disabling and fatal disease which is caused (in southern Africa) by the parasite *Trypanosoma brucei rhodesiense,* and is transmitted by the tsetse fly from the blood of vertebrate hosts on which the fly feeds						
Protozoa	Vector (biological - Tsetse fly)	Humans: If untreated considered fatal. If only treated after first stage, can result in significant and permanent neurological degeneration. *Animal (Mostly livestock):* Varies by species of animal and strain of protozoan parasite but generally causes loss of condition, and fatal if untreated in many cases.	Enzootic and endemic in animal and human populations, respectively.	Sylvatic pathogen	1 to 2 weeks (Humans) 4 days to 8 weeks (Domestic animals)	Much insecticide spraying, minimize contact with tsetse flies (netting)

and in-contact animals, exacerbating economic losses for farmers (Thomson et al. 2003). In the areas surrounding Kruger National Park, for example, more than 15,000 cattle were slaughtered following an FMD outbreak in 2001/2002 (Thompson et al. 2003). This outbreak happened as a result of livestock and wild animal contact after a flood knocked down a fence, highlighting how broader-scale processes can affect social-ecological function. However, the more common management strategy for FMD outbreaks is to contain animal movement from an infected area and to carry out extensive vaccination programs (Cumming 2011*b*), a practice that is strongly reflected in the region's dominant FMD management policies. Effects from both the disease and subsequent management actions will hugely influence local community perceptions of protected areas. In protected areas with buffalo (like Moremi National Park in Botswana or Hwange National Park in Zimbabwe), political pressures arising from outbreaks in the past have resulted in the development of new legislation that requires game-proof fences around protected areas to restrict movement of cloven-hoofed animals (Scoones et al. 2008, Cumming et al. 2015*b*). FMD has thus altered the built environment of protected areas and indirectly decreased revenues because of elevated costs associated with issuing permits, surveillance, assurance systems, vaccinations, and erecting fences (Scoones et al. 2008). For example, in Namibia, cattle farmers around protected areas like Bwabwata and Mangetti National parks are not able to export their beef (Scoones et al. 2008). On the other side, protected areas like Etosha National Park are unable to market themselves as "big five" destinations because game control policies dictate they remain free of buffalo. In terms of innovation, FMD has led to increased appreciation for the relevance of studies that track the movement behavior of the free-living buffalo that are reservoirs of the FMD virus (Ryan et al. 2006).

Anthrax is caused by the gram-positive, rod-shaped, spore-forming bacterium *Bacillus anthracis*. Spores lie dormant for decades (Hugh-Jones and de Vos 2002) and mildly infected wildlife can build an immunity against the disease (Pienaar 1961), hence contributing to ecological memory. The disease can become epizootic in both wild and domestic herbivores, which are infected through ingestion, inhalation, or absorption of spores, leading to abortions and adult mortality (Beyer and Turnbull, 2009). Disease outbreaks can move through the food web and are perceived to be part of natural ecosystem processes (Pienaar 1961, Hugh-Jones and Vos 2002). However, the disease poses threats to livestock in surrounding communities (Hugh-Jones and Vos 2002) and can spread to humans by direct contact or through consumption of infected animal tissue (Turner 1980). In Zambia, for example, 44 cases of anthrax were reported from people in areas surrounding North Luangwa National Park, evidently after eating meat from hippos (*Hippopotamus amphibius*; Hang'ombe et al. 2012). Management of the disease takes the form of carcass disposal by burning, burying, and decontamination, vegetation burning and barricading contaminated water sources (Pienaar 1961). Local perceptions of the protected area may therefore be influenced both by exposure to the disease and the regulations put in place to stop the spread of the disease, as was the case in the Zambian outbreaks (Siamudaala et al. 2006, Hang'ombe et al. 2012). The visual impact of carcasses, as well as the perceived link between the disease and the risk imposed on human health, may also affect visitor perceptions (as was the case in South Luangwa National Park, see Siamudaala 2005), visitation rates, and thereby revenue. The disease has caused social adaptation in the form of large investments in research and immunization programs, best evident in reserves where outbreaks are common, such as Mana Pools National Park, Etosha National Park, and Kruger National Park (Turnbull 1991, Beyer and Turnbull 2009).

The malaria parasite *Plasmodium* is transmitted from one human to another by the female *Anopheles* mosquito when it takes a blood meal as a prelude to the reproductive process. The disease does not affect wildlife or livestock, but can result in human mortality, and therefore has great implications for visitor perceptions. Being declared a malarial area, as is the case for most protected areas in northern South Africa, northern Namibia, Botswana, Zambia, Zimbabwe, and Mozambique, can cause drawbacks in visitor numbers and hence may indirectly reduce payments and revenue (Durrheim et al. 2001, Sachs and Malaney 2002) that are necessary for the protected area's persistence. In South Africa's KwaZulu-Natal province, for example, Durrheim et al. (2001) show that visitor numbers were significantly affected in iSimangaliso Wetland Park and Hluhluwe Umfolozi Nature Reserve following a malaria outbreak. Social adaptation has been evident in strategies to manage the disease through controlling mosquito numbers, e.g., spraying of dwellings with pesticides and reducing stagnant water areas, as well as preventing disease

Table 4. Potential effects of disease on elements of protected area identity. NP, national park; GR, game reserve, NR, nature reserve.

Disease Protected Areas	Effect on Components	Effect on Relationships	Effect on Innovation	Effect on Continuity
Foot and Mouth Disease (FMD)				
Moremi, Central Kalahari, Khutse GRs (Botswana); Bwabwata, Mangetti, Etosha NPs (Namibia); Kruger NP (South Africa); Hwange NP (Zimbabwe)	Domestic animals (cloven-hooved animal mortalities and slaughters) Built environment (fences) Revenue (cost of management)	Community perceptions (negatively affected because of domestic animal slaughter) Political pressures (for legislation to control FMD) Management (fence erection, animal slaughter)	Social adaptation (research into buffalo movements)	Ecological memory (virus maintained in buffalo) Legislation (designated FMD-free areas, compulsory slaughter of exposed animals)
Anthrax				
Etosha NP (Namibia); Kruger, Vaalbos NPs (South Africa); North and South Luangwa NPs (Zambia); Mana Pools NP (Zimbabwe)	Domestic animals (livestock, dog mortalities) Local communities (illness and mortalities) Visitors (reduced numbers) Revenue (reduction in tourist payments, cost of management)	Community perceptions (negatively affected because of human and domestic animal mortality) Visitor perceptions (negatively affected because of aesthetics of seeing carcasses and the association with bioterrorism) Payments (reduced visitor rates) Management (carcass disposal)	Social adaptation (research into immunizations)	Ecological memory (spores remain in ecosystem)
Malaria				
Moremi GR (Botswana); Gorongosa NP (Mozambique); Hluhluwe Umfolozi NP , iSimangaliso, Kruger NP (South Africa); Hlane GR (Swaziland); Blue Lagoon, Kafue, Lochinvar NPs (Zambia); Hwange NP (Zimbabwe)	Visitors (reduced) Revenue (reduction in tourist payments, cost of management)	Visitor perceptions (influenced by perceived risk of contracting the disease) Payments (reduced visitor rates) Management (spraying, reducing stagnant water)	Social adaptation (types of control and prevention)	Social memory (classified as malaria area)
Rabies				
Etosha NP (Namibia); Madikwe NR (South Africa); Hwange NP (Zimbabwe)	Wildlife (carnivora mortalities) Domestic animals (dog mortalities) Local communities (mortalities) Visitors (reduced numbers) Revenue (reduction in tourism, cost of vaccinations)	Ecosystem processes (trophic cascades) Visitor perceptions (reduction in charismatic species) Payments (reduced visitor rates) Management (potential vaccination of wild dogs)	Biological adaptation (hampered through loss of genetic diversity) Social adaptation (research into immunizations)	Conservation targets (reduced protection of endangered carnivores) Ecological memory (loss of genetic diversity)
Rift Valley Fever (RVF)				
Kalahari Gemsbok NP (Namibia); Limpopo NP (Mozambique), Addo, Kruger NPs (South Africa); Gonarezhou, Hwange NPs (Zimbabwe)	Wildlife (ruminant mortalities) Livestock (ruminant mortalities) Visitors (reduced numbers) Revenue (reduction in tourist payments)	Community perceptions (negatively affected because of humans and domestic animal mortality) Visitor perceptions (negatively affected because of risk of illness or mortality) Payments (reduced visitor rates)		Ecological memory (virus maintained in protected areas)

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Canine Distemper (CD)				
Chobe GR (Botswana), Etosha NP (Namibia)	Wildlife (carnivora mortalities) Domestic animals (dogs) Visitors (reduced numbers) Revenue (reduction in tourist payments)	Ecosystem processes (trophic cascades) Visitor perceptions (reduction in charismatic species) Payments (reduced visitor rates)	Biological adaptation (hampered through loss of genetic diversity)	Conservation targets (reduced protection of carnivores)
Trypanosomisasis				
Hluhluwe Umfolozi NP (South Africa); North and South Luangwa NPs (Zambia); Gonarezhou, Mana Pools, Matusadona NPs (Zimbabwe)	Domestic animals (livestock) Local communities (illness and mortalities) Visitors (reduced numbers) Revenue (reduction in tourist payments, cost of management)	Community perceptions (negatively affected because of human and domestic animal mortality) Social pressures (to control outbreaks) Visitor perceptions (negatively affected because of risk of illness or mortality) Payments (reduced visitor rates) Management (insecticide spraying)		

contraction, e.g., use of nets, repellents, and suppressive or causal prophylactics (O'Meara et al. 2010). Interestingly, in some areas, like Kafue, Lochinvar, and Blue Lagoon National Parks in Zambia, social adaptation has also taken the form of using government-supplied mosquito nets for other purposes, such as fishing, causing negative impacts on adjacent ecological communities (Haller and Merten 2008)

Rabies is a viral zoonosis caused by a negative-stranded RNA. It is acute, progressive, incurable viral encephalitis. Rabies can infect and be transmitted by a wide range of mammals, predominantly within the orders Carnivora (dogs, cats, jackals) and Chiroptera (bats; Lembo et al. 2010). Rabies can therefore directly influence wildlife both inside and outside protected areas. In the 1960s, domestic dogs were identified as the most likely source of rabies outbreaks in the Hwange National Park, which caused widespread mortality of endangered African wild dogs (*Lycaon pictus*; Childes 1988), highlighting the potential influence of domestic animals on protected area resilience. Similarly, canine rabies from domestic dog populations has been recognized as a major threat to the wild dog, which is a major attraction in South African National Parks (Hofmeyr et al. 2000, 2004, Lembo et al. 2010). Rabies could directly impact wild dog genetic diversity by reducing already low population numbers (Kat et al. 1995) and, in a broader sense, impact the endangered animal conservation targets of protected areas. Attempts to reintroduce wild dogs in Etosha National Park, where they would be safe from the risks they face on surrounding agricultural land, have all been thwarted, mostly as a result of rabies-related die-offs (Scheepers and Venzke 1995). Through changes in carnivore abundance in protected areas, the rabies virus has the potential to disrupt vital ecosystem processes (Estes et al. 2011). Vaccination is a possible, yet costly management action, as is evident from studies in the Madikwe Nature Reserve (Vial et al. 2006).

Protected areas are thought to act as reservoirs of RVF in southern Africa (Olive et al. 2012), making this disease an important factor in the ecological memory of protected areas. RVF is a contact and mosquito-borne disease caused by the RVF virus, an enveloped RNA segmented virus in the family *Bunyaviridae*, genus *Phlebovirus*. Infection in ruminants can result in spontaneous abortion of fetuses, or death (Swanepoel and Paweska 2011). Humans contract the disease via contact with tissues of infected animals or via mosquito bite, which usually results in an influenza-like illness, but substantial human mortalities have occurred during large outbreaks (Swanepoel and Paweska 2011). The disease can thus affect human, wildlife, and livestock components of protected area identity directly during outbreaks, as have been recorded in South Africa, Zimbabwe, Namibia, and Mozambique (Swanepoel and Paweska 2011). In particular, the extensive livestock losses that resulted from these outbreaks have resulted in severe economic losses (Swanepoel and Paweska 2011), and in some cases have led to negative local community perceptions toward protected areas. Visitor perceptions and revenue from visitors may also be affected: based on a (incorrectly) suspected case of RVF in a German tourist following a trip to a South African game reserve, the World Health Organization issued travel advisories to tourists attending the 2010 FIFA World Cup (Boshra et al. 2011).

CD can have a significant impact on predator populations within a protected area. The virus is transmitted via aerosol droplets, can remain virulent in standing water, is highly contagious and deadly, and is known to affect a variety of species in the order of Carnivora (Alexander et al. 1996, Roelke-Parker et al. 1996). Outbreaks of CD have had devastating effects on lion (*Panthera leo*), bat-eared fox (*Otocyon megalotis*), spotted hyena (*Crocuta crocuta*), and wild dog populations in southern African protected areas such as Etosha National Park and Chobe Game Reserve (Alexander et al. 1996, Roelke-Parker et al. 1996, Bellan et al. 2012). The prevalence of CD in domestic dogs found in communities surrounding protected areas provides a constant disease reservoir (Alexander et al. 1996), making CD important for ecological memory. Reduced population sizes of top predators negatively impacts biological adaptive potential by decreasing genetic diversity. As with rabies, CD can disrupt ecosystem processes through trophic cascades and affect large carnivore

population sizes. Charismatic species are important drawcards for tourists and large carnivore losses may therefore impact visitation rates (Lindsey et al. 2007).

Trypanosomiasis of humans in east and southern Africa is a disabling and fatal disease caused by *Trypanosoma brucei rhodesiense* (Sistrom et al. 2014) that is transmitted by tsetse flies that feed exclusively on vertebrate blood. In southern Africa, several cases of human sleeping sickness have occurred recently in protected areas within the Zambezi Valley in Zimbabwe (WHO 2015), and the disease is common in the Lambwe Valley of Kenya (Wellde et al. 1989). Primary wild hosts of tsetse and reservoirs of trypanosomes in southern Africa are warthog, bushpig (*Potamochoerus larvatus*), kudu, bushbuck, buffalo, and elephant (*Loxodonta africana*; Hargrove 2003). *T.b. brucei* is primarily responsible for trypanosomiasis or nagana of domestic livestock where these occur within tsetse areas or adjacent to protected areas harboring tsetse. Outbreaks in livestock in areas surrounding protected areas are common: examples include a 1990 outbreak in Hluhluwe Umfolozi National Park, South Africa (Kappmeier et al. 1998) and several outbreaks around the Luangwa National Park in Zambia (Anderson 2009). Such outbreaks impose a major burden on the rural poor, by impacting both people and their livestock (Wellde et al. 1989, Welburn et al. 2006, Anderson 2009, Torr et al. 2012). Management to date has been largely reactive, including game elimination and widespread insecticide spraying (Welburn et al. 2006, Cumming et al. 2015*b*) that may negatively impact biotic and abiotic components of protected area identity. For example, tourists visiting protected areas in east and southern Africa have contracted Rhodesian human trypanosomosis (Simarro et al. 2012) and outbreaks are likely to affect tourist visitation rates. In terms of innovation, wildlife in areas still affected by the disease seems to develop a partial immunity to the disease. In Matusadona National Park, introduced white rhinos (*Ceratotherium simum*), from populations that had not been exposed to the disease since 1948, died after contracting sleeping sickness, although black rhinos (*Diceros bicornis*) endemic to the region did not become diseased (Taylor 1986).

As these examples show, pathogens, and the diseases that they cause, have the potential to significantly alter the identity of a protected area. As noted already, alteration of the biotic community in a protected area can impact other elements of identity, with potentially important economic, social, and ecological consequences. We have chosen our disease case studies to be representative of the different ways in which protected area identity could be impacted by a disease outbreak, as documented in the literature and by veterinary experts (Table 4; Appendix 1). Disease may enter a system in different ways and have cascading impacts that affect a range of different elements of protected area identity.

DISCUSSION

Components of identity that were most pertinently affected in our case-studies, both directly and indirectly, were wildlife, domestic animals, local communities, visitors, and revenue. Revenue was a component that was consistently affected across case-study diseases. Revenue loss occurred either because of the direct costs involved in disease prevention, e.g., in FMD and Anthrax, or as a result of negatively affected tourist perceptions, e.g., fear of malaria. Tourist perceptions seem most affected by diseases that affect people directly, and to a lesser extent by diseases that only occur in animals. Conversely, successful management of disease, as in tuberculosis-free or malaria-free zones, might result in additional revenue from tourists willing to pay a premium for parks with these assets/benefits.

Another general pattern evident from our case studies is that pathogens entering the protected area system via domestic animals can have dire ecological consequences. These consequences often result from the effect of disease on the relationships between components. For example, both rabies and CD result in mortalities and disease in top carnivores, which disrupts ecological function via trophic cascades. Thus, one of the major influences on wildlife management in southern Africa is that of the interactions between wildlife and domestic animals in surrounding areas. Concerns about pathogen and parasite transmission from wildlife to domestic analogues, e.g., FMD, bovine tuberculosis, and tick-borne pathogens between buffalo and cattle, are a major concern for both small-scale farmers and meat exporters. Exchanges between wild and domestic ungulates tend to be of greatest socioeconomic concern when they move from wildlife to domestic stock, because many wildlife species are natural reservoirs for pathogens and do not become clinically ill when infected. However, removing buffalo from areas of high buffalo/cattle coincidence and contact may not necessarily improve protected area resilience to disease outbreak. In addition to the potential negative economic consequences from tourist-related income loss (Thomson 2009), ecological feedbacks from the removal of grazing mammals may actually increase the likelihood of a disease outbreak. In long-term exclosure experiments, Kenya, Keesing and Young (2014) found that a removal of grazing mammals resulted in an increase in rodent abundance, which devastated acacia seedlings, attracted more venomous snakes, and doubled the abundance of fleas, so increasing the risk of transmission of flea-borne pathogens.

Management decisions about disease control, particularly when diseases are introduced via domestic animals, may produce profound ecological feedbacks, as is also evident in many protected areas outside our study area. Using discrete time models fitted to population time-series data, Holdo et al. (2009) have showed how suppression of endemic rinderpest in the wildebeest population caused the Serengeti ecosystem to increase its woody component by a factor of two to three, consequently facilitating its switch from a net carbon source to a net carbon sink over a 40-year period. Further examples of social processes producing ecological feedbacks with social consequences come from the ecohealth and landscape epidemiology literatures, with many studies now drawing the link between landscape degradation, pathogen spread, and negative consequences for human health and well-being (see Myers et al. 2013, Berbés-Blázquez et al. 2014 for examples).

Ecosystems have evolved with pathogens and parasites as system elements, and our case study examples demonstrate that ecological communities are often quite resilient to endemic pathogens. Diseases that are endemic to protected areas are likely to affect resilience predominantly through social and economic pathways. In our case-studies, for example, trypanosomiasis outbreaks impacted people and livelihoods, rather than affecting

ecological components directly. Similarly, control methods such as fences, game elimination, and DDT spraying have had major ecological effects, such as disrupting migratory pathways of large-ranged species and causing population declines of top predators (Gadd 2012, Cumming et al. 2015*b*). In many ways, the human social and economic components of protected areas and their surroundings have greater potential than the ecosystems themselves to be affected by endemic disease, and to impact the resilience of a protected area to a disease outbreak.

Diseases also affect elements of innovation and continuity in protected areas. In cases where wildlife pathogens are zoonotic, i.e., can be transmitted to people, pathogens may have long-term effects on both the establishment and the maintenance of protected areas. Trypanosomiasis and human malaria are both perceived as low-altitude diseases, and both have had marked effects on settlement patterns in Africa. Many existing protected areas in southern Africa were originally proclaimed as hunting areas because of their combination of erratic rainfall, poor soils, and disease (Cumming and Atkinson 2012). As a result, the habitats of greatest conservation concern are currently highveld grasslands and woodlands, rather than lowland savannas. Pathogens such as malaria continue to influence tourism and a potential tsetse fly range expansion in a changing climate could have similarly significant impacts.

Additionally, our CD and RVF case studies show how ecological memory is affected through at least two pathways: the loss of genetic diversity and persistence of the pathogen in the environment, e.g., RVF in mosquito eggs. More positively, social adaptation can lead to more insightful management or more research on diseases. However, even social adaptation can express itself in undesired and unintended ways, such as the Mozambican example of local communities reportedly using malaria nets as fishing gear (Blythe et al. 2013).

Although pathogens may have a number of direct effects, particularly on domestic stock, endemic pathogens often cause perturbations to social-ecological systems through indirect effects. From our case studies it appears that, although most pathogens found in protected areas do not kill large numbers of wild animals or infect many people, occasional outbreaks and mortalities have a large impact on public perceptions and management decisions. Management responses and the indirect socioeconomic effects resulting from pathogen outbreaks are not always rational or proportional to the actual severity of the threat (Stirling and Scoones 2009).

The many complexities raised by pathogens in complex systems suggest a strong need for social-ecological systems approaches to landscape epidemiology (Cumming et al. 2015*c*). Highlighting the indirect, cascading effects that are generated by cross-scale feedbacks and may not be detected by deterministic and linear approaches, Palomo et al. (2014) have considered how systems thinking and social-ecological theory may be useful to the management of protected areas for disease outbreaks. Their paper also represents a primary juncture of convergence for the resilience, ecohealth, and landscape epidemiology literature. Berbés-Blázquez et al. (2014) suggest that the ecohealth paradigm can benefit from resilience thinking by a focus on practical management tools, such as adaptive environmental management; building on synthetic concepts such as scale interactions; and an integration of panarchy and regime shifts concepts to add a more dynamic perspective to ecohealth. Similarly, Meentemeyer et al. (2012) suggest that an explicit consideration of reciprocal feedbacks between human activities and disease dynamics offers potential insight about the tipping points between successful collective action and unmanageable invasion.

We have focused primarily on ways in which pathogen-driven perturbations may decrease system resilience by eroding or changing elements of system identity. However, it is important to note that pathogens and parasites may also have positive influences on the resilience of ecological communities. They reduce crowding in wildlife populations and may enhance their genetic fitness by removing sick and old animals, and/or making them easier prey for predators. It is axiomatic in parasite ecology that a very small proportion of the animals in a population carry a very large proportion of its parasites (Poulin 2007). The numeric fluctuations that pathogens can introduce into wildlife populations may further enhance ecosystem resilience by creating spatial and temporal heterogeneity that helps to maintain diversity in both species and habitat composition within a protected area.

Pathogens therefore form an integral part of the identity of protected areas (Castello et al. 1995, Ayres and Lombardero 2000). The presence of endemic diseases or pathogens in the system may influence the functioning of protected areas, directly or indirectly, and with either positive or negative consequences. Pathogen outbreaks differ from other types of disturbances like fire, floods, and political and economic downturns in the sense that they are often produced or exaggerated by intrinsic factors. Understanding how disease affects the resilience of protected areas is of critical importance for understanding the overall resilience of protected areas. Disturbance forms an integral part of development because it opens up opportunities in terms of recombination of processes, system renewal, and emergence of new trajectories (Folke 2006). There is only a finite amount of disturbance that a system can absorb, however, before it shifts into a different state (Holling 1973), and the dynamics after a disturbance are critically dependent on the self-organizing capacity of the system (Norberg and Cumming 2008). Pathogens provide sources of adaptation, innovation, and continuity to protected areas, and the emergence or outbreak of a disease in a protected area may spur new research, lead to collaboration with local communities, and provide motivation for the development of better vaccination or control strategies. As pathogens affect resilience in a diversity of ways, they also offer an opportunity to advance social-ecological theory through more rigorous comparative analyses of case studies. Although the complexity of the problem does not permit the development of simple heuristics for understanding the influence of pathogens on protected area resilience, it is clear that the impacts of pathogens are social-ecological in nature and should ideally be approached from an integrated, social-ecological systems perspective.

Responses to this article can be read online at:
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Appendix 1

Table A1.1. This table shows examples of disease outbreaks in southern African protected areas for each of the case study diseases considered in this study. For each case study disease, we give the protected area(s) where the focal disease had an impact, as well as a short description of what this impact was. “NP” or “NPs” denote a National Park and National Parks, respectively and “GR” denotes a Game Reserve.

Disease	Protected Area	Disease Outbreak	Impact	Reference
Foot and Mouth Disease	Kruger NP, South Africa	In 2001/2002 heavy floods resulted in the destruction of a fence, cloven-hooved carriers came into contact with domestic stock as a result.	Significant production losses in domestic stock when more than 15,000 cattle were slaughtered	Thomson et al. 2003
	Moremi NP, Botswana, Hwange NP, Zimbabwe (examples)	Several outbreaks of various magnitudes in the earlier part of the 1900s	Development of new legislation that requires game-proof fences around protected areas, as well as other infrastructural and permitting measures to restrict movement of cloven-hoofed animals.	See Cumming et al. 2015a for a history of policies, Vosloo et al. 2002 for a description of different outbreaks, Schoones et al. 2008.

	Bwabwata, Mangetti and Etosha NPs, Namibia; Central Kalahari & Khutse GRs, Botswana	Several outbreaks of various magnitudes in the earlier part of the 1900s	Farmers around protected areas with bufallo behind the "red line" are not able to export their beef. Areas south of the red line are unable to market themselves as “big five” destinations, as the game control fences dictate they remain free of buffalo.	Schoones et al. 2008
	Kruger NP, South Africa	Several outbreaks of various magnitudes in the earlier part of the 1900s	Increased appreciation for the relevance of studies that track the movement behaviour of the free-living buffalo that are reservoirs of the FMD virus.	e.g. Ryan et al. 2006
Anthrax	Etosha NP, Namibia; North and South Luangwa NPs, Zambia, Vaalbos, Kruger NPs, South Africa	Several outbreaks of various magnitudes between 1900 and 2011	Infection of livestock and humans in surrounding communities through direct contact or through consumption of infected animal tissue, leading to illness and death.	Hugh-Jones and Vos 2002, Hang'ombe et al. 2012, Simudaala et al. 2006

North Luangwa NP, Zambia	Outbreak in 2011, Incidences of Anthrax in 1990, 1991-1998.	Local perceptions of the protected areas, and eating meat from it are affected by both Anthrax outbreaks and enforcement of regulatory control measures of the disease.	Hang'ombe et al. 2012, Siamudaala et al. 2006
South Luangwa NP, Zambia	Outbreak in 1987, 1997	Tourist lodges in South Luangwa National Park are located along the river where anthrax deaths are common. Delay in disposing off anthrax carcasses around tourist centres such as lodges and campsites exposed tourists to the unpleasant and offending odour from the decomposing carcasses. Burning of carcasses within lodge premises exposed tourists to smoke pollution. The presence of carcasses along game viewing roads compromised the aesthetic beauty of the area and caused concern among the tourists.	Siamudaala et al. 2005
Mana Pools NP,, Zimbabwe; Etosha NP, Namibia, Kruger NP, South Africa	Several outbreaks of various magnitudes between 1900 and 2008	Social adaptations in the form of large investments in research, resulting in immunization programmes.	Turbull 1991, Beyer and Turnbull 2009

Malaria	Kruger NP South Africa, Gorongosa NP, Mozambique, Hwange NP, Zimbabwe, Moremi NP, Botswana, Etosha NP, Namibia, Kafue NP, Zambia	Presence of risk to contract Malaria	Being declared a malaria area affects the continuity of an affected protected area as it becomes embedded into the system's memory.	Durrheim et al. 2001
	IsiMangaliso Wetland Park and Hluwe-Imfolozi GR, South Africa	1997 Malaria Outbreak	An outbreak of malaria decreased visitation rates to IsMangaliso and Hluwe-Imfolozi Nature reserves	Durrheim et al. 1998
	Kruger NP, South Africa	Presence of risk to contract Malaria	Extensive press coverage of South African epidemics, including malaria, appear to have negatively influenced the volume of tourism to the Kruger National Park	Durrheim et al. 2001

	Kafue-, Lochinvar- and Blue Lagoon NPs, Zambia	Presence of risk to contract Malaria	social adaptation has also taken to form of using government-supplied mosquito nets for other purposes, such as fishing, causing negative impacts on adjacent ecological communities	Haller & Merten 2008
	Hlane Game Sanctuary, Swaziland (example - many others)	Presence of risk to contract Malaria	Social adaptation has been evident in strategies to manage the disease through controlling mosquito numbers (e.g. spraying of dwellings with pesticides, reducing stagnant water areas) as well as preventing disease contraction (e.g. use of nets, repellents and suppressive or causal prophylactics)	O'Meara et al. 2010, Hackel & Carruthers 1993
Rabies	Hwange NP, Zimbabwe	1966 Outbreak	In the 1960s, domestic dogs were identified as the most likely source of rabies outbreaks in the Hwange National Park, which caused widespread mortality of wild dogs (Childes 1988), highlighting the potential influence of domestic animals on PA resilience	Childes 1988

	Etosha NP, Namibia; Madikwe GR, South Africa	1978, 1989, 1990 (Etosha); 1998 and 2002 (Madikwe)	Rabies have thwarted attempts to re-introduce endangered wild dogs in Etosha National Park, has resulted in two successive outbreaks in wild dog packs in Madikwe, South Africa (Hofmeyr et al., 2000 and Hofmeyr et al., 2004), and has been identified as the cause of the loss of five packs in Botswana (Woodroffe et al., 2004).	Hofmeyr et al. 2000, 2004, Scheepers and Ventske 1995
	Madikwe GR, South Africa	Following oubreaks in the 1990s	Multiple doses of expensive vaccines were needed to succesfully protect wild dog pups from Rabies	Vial et al. 2006
Rift Valley Fever	Hwange NP, Zimbabwe; Kruger NP, South Africa	Several outbreaks in the 20th century	Protected areas are thought to act as reservoirs of RVF in several southern African Protected areas	Olive et al. 2012

Kalahari Gemsbok National Park, Namibia; Addo NP, South Africa	1975 Outbreak in South Africa	Human mortalities and illnesses have occurred during large outbreaks	Swanepoel and Paweska 2011
Kalahari Gemsbok NP, Namibia and Addo NPs, South Africa	1955, 1969, 1970, 1974-1976 Outbreaks	Extensive livestock losses (Angora goats and Cattle) led to decreased revenue and community perception	Swanepoel and Paweska 2012
Addo NP, South Africa	Previous recorder outbreaks of RVF (invluding one in 2010)	Travel advisory issue after suspected case of Rift Valley fever	Boshra et al. 2011

Canine Distemper	Chobe GR, Botswana, Etosha NP, Namibia	Several Outbreaks in the 1900s and early 2000s	Outbreaks of CDV have had devastating effects on lion, bat eared fox, hyena and wild dog populations in Southern African protected areas	Alexander et al. 1996, Bellan et al. 2012
	Chobe GR, Botswana	1994 Outbreak	The prevalence of CDV in domestic dogs found in communities surrounding protected areas provides a constant disease reservoir (Alexander et al. 1996), making the disease important for ecological memory.	Alexander et al. 1996
Trypanosomisasis	Gonarezhou NP, Zimbabwe (example)	With reference to eradication measures in the 1920s	Game elimination and widespread insecticide spraying have negatively impacted biotic and abiotic components of PA identity.	Cumming et al. 2015a, Welburn et al. 2006

North and South Luangwa NPs, Zambia and Mana Pools NP, Zimbabwe	Several recorded cases in the 1900s and 2000s	Tourists visiting protecting areas in East and southern Africa have contracted Rhodesian human trypanosomosis; outbreaks are likely to affect tourist visitation rates to the affected protected areas.	Simarro et al. 2012
Hluhluwe Imfolozi NR, South Africa, South Luangwa NP, Zambia	Outbreaks in the 1980s and 1990s	Outbreaks of Trypanosomiasis results in losses of livestock and represent a major disservice to the rural poor	Kappmeier et al. 1998, Van de Bosch et al. 2006, Welde et al. 1989, Torr et al. 2012, Anderson et al. 2009
Matusadona NP, Zimbabwe	With reference to a reintroduction in 1984	Rhinos relocated died from Trypanosomiasis despite local populations not becoming diseased	Taylor 1986