

The Impact of Protected Areas on the Incidence of Infectious Diseases

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Abstract

Background: The natural environment provides multiple ecosystem services, and thus welfare benefits. In particular, it is known that different ecosystems, such as forests, contribute to human health through different ecological interactions, and that degradation of these natural ecosystems have been linked to the emergence and re-emergence of infectious diseases. However, there is little evidence on how ecosystem conservation policies affect human health. In Chile, about 20% of national land is under protection by its national network of public protected areas.

Methods: We use a database of mandatory reporting of diseases between 1999 and 2014, and considering socio-economic, demographic, climate and land-use factors to test for a causal relationship between protected areas and incidence of infectious diseases using negative binomial random effects models.

Results: We find statistically significant effects of protected areas on a lower incidence of Paratyphoid and Typhoid Fever, Echinococcosis, Trichinosis and Anthrax.

Conclusions: These results open the discussion about both causal mechanisms that link ecosystem protection with the ecology of these diseases and impacts of protected areas on further human health indicators.

JEL Codes: Q58, Q57, Q56, Q01

1. Background

Humans have caused significant losses of natural resources and deterioration in natural ecosystems through technological changes and cultural activities such as the use of irrigation systems, intensive exploitation of water reserves and land for food production, logging, among others. [1]

This human interaction has led to a degradation or unsustainable use of 60% of the planet's ecosystem services, including water and air purification, local and global climate regulation, plagues, and natural hazards. [2] These alterations to ecosystems have occurred faster in the last 50 years than in all the rest of the history of humankind. Furthermore, the drivers behind these changes do not seem to decrease, but in many cases only increase in intensity. [3]

At the same time, during the last century little attention has been paid to how these changes in the structure of natural systems and their functions may affect human health. [4] The causal relationships between ecosystem change and human health are complex since they are mostly indirect, scattered in time and space, and dependent at the same time on other factors. [1]

Nonetheless, there is growing evidence on how environmental changes, at different levels, can produce impacts on human health, such as stress, respiratory allergens, infectious diseases, water shortages and associated health problems, food insecurity, health issues associated to population displacement due to environmental changes, health problems associated with air pollution, among many others. [4]

It should be emphasized among the examples shown previously, that the emergence and re-emergence of infectious diseases has become a global environmental problem with major consequences, both in public health, as in economics and politics. The etiological origin of most emerging diseases is zoonotic (i.e., infectious

diseases caused by bacteria, viruses and parasites that spread between animals (usually vertebrates) and humans), and the environmental change that affects the habitat of wildlife has been highly involved in the onset of these diseases and their spread. [5]

Currently, unprecedented environmental changes are taking place that will affect the lives of human beings and animals with whom they interact. [6] These environmental changes are not only related to climate, but also to demographics, changes in land use and environmental conditions at small and large scale. [7] These changes are altering the interactions between people and infectious diseases, in addition to influencing vectors of zoonotic parasites and their relationships with humans. [6] At the same time, land use changes in the form of deforestation, are considered one of the most important environmental factors driving the appearance of emerging and re-emerging of infectious diseases. [8] [9] Nonetheless, much of the literature and experimental studies on forests and human health has been carried out in tropical areas because diseases are less prevalent in temperate climates, due to the occurrence of a cold season.[10]

In terms of the impact of protected areas on human health, there is little evidence on how forest and ecosystem conservation policies affect human health. [11] Nevertheless, it is estimated that between 23% and 25% of global morbidity could be avoided by improving management of environmental conditions. [12] In this direction, although conservation is the main purpose of these projects, the sense of linking protected areas with other components of development and welfare, such as health, becomes evident. [13]

Outbreaks of emerging diseases from zoonotic origin are affecting the human population at an alarming rate [14]. In this regard, in recent decades the number of emerging pathogens that affect human population have substantially increased. [15] Of these infectious diseases, 62% are zoonotic. [16] Knowledge of the relationships between host, infectious agents and environment is crucial to counter pathogens, especially zoonotic diseases. This is critical to better understand the reach and limits of the current SARS-CoV-2 pandemic, which report reaches by September 2020 around 32 million confirmed cases worldwide after emerging in China at the end of 2019 [17]. Human CoV infection is a zoonosis and the SARS-CoV-2 experience has shown us how devastating and life-threatening a zoonotic disease could be. Therefore, understanding the role of conservation policies in the transmissibility of infectious diseases would provide valuable information to better plan and design future conservation actions.

Ecological factors associated with the appearance of these diseases are complex and poorly understood. [14] The main drivers of the onset of a disease include the exponential population growth, consumption and waste generation. This in turn has prompted the intensification and expansion of urbanization and agriculture, and forest habitat alteration, which determines the environmental change in a region. The process of occurrence of infectious diseases is associated with the combination of these environmental factors.

In addition, a common element involved in outbreaks of emerging diseases, is always the sudden social and ecological disruption, reflected in changes in land use. [18] Changes in disease vectors and reservoirs domestication, domestic habitat invasion by opportunistic species, and invasion of natural habitat for wild species are associated to these trends of land use changes. The species become receptors of pathogens in altered and fragmented forests near human settlements. The interaction between human and animal hosts, and reservoirs and vector species within ecosystems affects the dynamic host-pathogen, facilitating the onset of the disease. [18]

Moreover, while there is plenty of evidence on the impact of the drivers that promote ecosystem change, the linkage between ecosystem change and benefits coming from ecosystem services has not been well documented. This contributes to the perception that conservation initiatives involve only costs to local population by constraining social benefits of extractive human uses. [19] [11]

The objective of this article is to measure the impact of the Chilean National Public System of Protected Wild Areas on outcomes of infectious diseases. To help this objective, we built a panel dataset containing information on mandatory notification of diseases, land use, biophysical conditions and demographic and socioeconomic factors. In addition, we summarize the existing evidence that propose how protected areas might affect the chosen diseases outcomes and calculate the marginal effects, showing how diverse levels of protection are linked to different impact on disease incidence.

a. Chilean System of Protected Areas

Continental Chile stretches more than 4200 km north to south, 445 km at its widest point and 90 km at its narrowest part. Its geography is characterized by the Andes and Coastal ranges flanking the so-called “Intermediate Depression”, interrupted on several occasions by regional variations. [20]

The climate varies greatly and comprises the world’s driest desert in the north, Mediterranean climate in the center, humid subtropical in Easter Island, oceanic climate, alpine tundra and glaciers in the east and south, among many other ecoregions. One of them, Valdivian temperate forest, is considered as one of the top biodiversity hotspots in the world. [21]

Chile established its first protected area in 1907 and the first national park in 1925, in a context of rapid deforestation and increased awareness about conservation. However, it was not until 1984 that the National System of Protected Wild Areas (SNASPE by its acronym in Spanish) was created to organize the protected areas in a unified system in order to preserve Chilean natural resources. It is regulated by law and administered by the Chilean Forest Service. [22]

At present, the system includes 41 national parks, 46 national reserves and 18 natural monuments that cover 21,1% of the continental Chilean territory. [23] 99,8% of the protected area is concentrated in the first two categories. [21] However, while most threatened ecosystems are located between central and south Chile, 80% of protected areas belong to the two southernmost regions where land has low commercial value and low population density. [21][22]

2. Methods

a. Data

Our dataset comprises 16 years (1999–2014) of annual municipal-level observations of mandatory reporting of cases of Echinococcosis, Trichinosis, Typhoid and Paratyphoid fever and Anthrax. Syphilis, Hepatitis B and Rubella cases were also included as negative controls.

The infectious diseases were chosen due to their importance in public health, differences in their infection cycle and pathogen, and presence in Chile. These characteristics will be discussed in the description of the health outcome to be analyzed. Syphilis, Hepatitis B and Rubella were included as negative controls because they are not theoretically related to landscape-level ecological changes.

The statistics are based on mandatory reporting of diagnosed cases, from both public and private sectors. The diagnosed cases are attributed to the municipality of residence of the patient and not to the municipality of the health facility. Municipality is the basic political and administrative unit in Chile.

In addition, we compiled municipality-level cross-sectional data for the percentage of municipal land covered by protected areas, population, road density, distance to region capital, municipality land area, and biophysical conditions, such as temperature, rainfall, altitude and slope. These data were collected for 336 of the 345 existing municipalities, spanning the entirety of the sixteen administrative regions of Chile.

Data on diseases were obtained from the Department of Health Statistics and Information of the Ministry of Health. Additional data were provided by the Ministry of Public Works, the Ministry of the Environment, National Statistics Institute and University of La Frontera.

Descriptive statistics for all dependent and independent variables are shown in Table 1 followed by a brief narrative of the description of the variables.

Table 1
Descriptive Statistics

	Mean Value Municipalities Without Protected Areas	Mean Value Municipalities With Protected Areas	Diff Mean Value	Norm diff ^a	t- statistic
(Para) Typhoid Fever (cases/year)	1.255	0.952	-0.304	-0.103	2.822
Echinococcosis (cases/year)	0.649	1.250	0.601	0.240	-9.978
Trichinellosis (cases/year)	0.111	0.102	-0.009	-0.010	0.131
Anthrax (cases/year)	0.009	0.011	0.002	0.015	-0.624
Syphilis (cases/year)	9.646	8.466	-1.180	-0.049	1.130
Hepatitis B (cases/year)	1.523	1.085	-0.437	-0.081	2.222
Rubella (cases/year)	1.646	0.788	-0.858	-0.084	2.497
Population	48061.470	34823.180	-13238.290	-0.203	5.766
Roads density (km/km ²)	0.766	0.366	-0.401	-0.878	26.348
Distance to regional capital (km)	61.122	100.393	39.272	0.645	-21.639
Slope (°)	7.657	12.125	4.468	0.801	-28.859
Altitude (masl)	617.527	897.616	280.089	0.345	-12.869
Average temperature in July (°C)	6.484	4.778	-1.706	-0.531	19.061
Average temperature in January (°C)	16.274	13.631	-2.643	-0.782	26.106
Rainfall (ml)	874.754	1170.980	296.226	0.447	-15.202
N municipalities with protected areas (treated) = 1600; N municipalities without any protected area (control) = 3820.					
$= \frac{\bar{x}_T - \bar{x}_C}{\sqrt{\frac{s_T^2 + s_C^2}{2}}}$ ^a Normalized difference where T= protected and C= unprotected.					

Treatment variable. This is the explanatory variable of interest or treatment variable and it is expressed as the proportion of the municipality land area covered by protected areas. There are 240 municipalities that have no protected areas established under the SNASPE system which will be used to estimate the counterfactual scenario. Among protected municipalities, the maximum proportion of protection is 72.7% for the case of the municipality of Juan Fernández.

Health outcomes. The first seven variables shown in Table 1 correspond to the dependent variables or health outcomes. They show the number of cases of diagnosed patients per municipality per year for each of the chosen mandatory infectious diseases. As shown in Table 1 and following the treatment variable definition, it can be seen that there are systematic differences between treated and control municipalities for almost all health outcomes as expressed by the normalized difference between treated and control municipalities.

Patterns of infectious diseases in humans are affected by economic conditions, changes in human behavior and environmental factors. All these driving factors correlated not only with health outcomes but also with the establishment of protected areas will also shown in Table 1 as co-variables. [24–26] All transmission of zoonotic diseases may be altered through changes in ecosystems. The physicochemical environment influences the pathogen and its survival, density and dispersal, the conditions that determine the survival of the vector, the development of the pathogen after the interaction with the vector, the presence, behavior and abundance of intermediate hosts and definitive hosts, and it even influences the nutritional status and behavior of humans. [27]

It is important to consider that there are different causal mechanisms by which environmental change can alter disease transmission, through impacts on pathogens, vectors or hosts. [27] These mechanisms include changes in exposure pathways, density or identity of disease-related organisms, species composition of communities of organisms, alteration in life cycle of pathogens or vectors and change in the environment in which organisms live, that creates genetic alterations, which increases transmission. [28] This research is not aiming to estimate these causal mechanisms, but to estimate the causal impact of protected areas on the incidence of some infectious diseases.

Echinococcosis

Echinococcosis caused by *Echinococcus granulosus* is the main cause of hospitalization for parasitic disease in Chile. It can produce high degrees of morbidity to people who are mostly in full productive age. [29]

Echinococcosis is a zoonotic disease caused by two species of cestode larvae, being *Echinococcus granulosus* the most common and widespread species. This parasite has different life cycle stages, and it depends on the dog-sheep cycle as definitive and intermediate host, respectively. Humans are aberrant hosts that can serve as definitive or intermediate host by ingesting eggs. Eggs release oncospheres in the intestine, which then develop hydatid cysts in a variety of organs. [30] Echinococcosis generates a high socio-economic impact due to days not worked, surgery and recovery, medication, medical visits and related medical services. In general, it is related to rural pastoralist, and the sheep is the intermediate host of greatest epidemiologic importance. Other animal species can also acquire relative importance, such as pigs, goats and South American camelids. [31]

In Chile the annual costs derived from surgical treatment to remove a hydatid cyst in 2012 was estimated in USD 2.46 million and USD 3.13 million summing the costs of sick leaves and loss of productivity respectively. These costs are only considering human costs. There are also animal-derived costs that considers pharmacological treatment of infected dogs and animal production losses derived from confiscations and reduction of meat production. Considering both animal and human costs, the annual economic burden of the disease in 2012 was estimated in USD 14.25 million. [29]

Trichinosis

Trichinosis or trichinellosis is a tissue-dwelling nematode infection. Tissue-dwelling nematode infections are widely distributed throughout the world and their impact is greatest in resource-poor settings in the tropics and subtropics, but they also affect population in temperate more developed regions, where they continue to be at risk. [32]

Trichinosis or trichinellosis is a zoonotic disease caused by eight species of the nematode *Trichinella spiralis* that has been found in more than 66 countries and 150 mammalian species, and it represents the most common species responsible for human trichinellosis. [33]

In Chile, human trichinosis is endemic and the first indigenous cases were published more than a hundred years ago and its occurrence is closely related to cultural habits and diet. [34] *T. spiralis* has been found in domestic pigs, rats, cats, dogs and pumas [35], from which pigs and rats have a greater importance in domestic infection cycles. Domestic pigs usually eat infected rats, causing trichinellosis in humans by the ingestion of undercooked meat containing encysted larvae. [29] After exposure to gastric acid, the larvae are released from the cysts and invade the small bowel mucosa where they develop into adult worms. [36]

Trichinosis is worldwide distributed and it has important socio-economic impacts in public health and local food production economies. [37] The epidemiology of trichinosis is under constant review. Worldwide it has been considered as a reemerging infection because new species have appeared, as a result of man having invaded other habitats thus exposing himself to new cycles of infection. [34]

Anthrax

Anthrax is also zoonotic, but the main difference with the previous two diseases is that this is virulent, contagious and potentially fatal. [38] Anthrax is caused by the bacteria *Bacillus anthracis*, which infects both, wild and domestic herbivores. Susceptibility to the disease is greatest for cattle followed by sheep, horses and goats that get infected by contaminated food or water. Infected herbivores generally bleed from the nose, mouth and bowel before dying; resulting in contamination of pastures and water sources. [39] In addition, *B. anthracis* can be disseminated by flies and mosquitos carrying contaminated feces or blood from infected herbivores. [38] In humans, infection can be cutaneous, gastrointestinal and inhalational and they occur by direct contact with infected animals or animal products. [40][41] Most anthrax cases are associated with industrial or agricultural workers. In Chile, references can be found prior to 1950 linked to slaughter of animals. Rural areas have shown higher lethality and occurrence [42].

Typhoid and paratyphoid fever

Typhoid and paratyphoid fever are infectious diseases caused by *Salmonella typhi* and *Salmonella paratyphi*, respectively. They are together known as enteric fever. Typhoid and paratyphoid fever are commonly related to the consumption of unsafe water and food. [43] Nevertheless, a range of other risk factors have also been identified, such as presence of water bodies, flooding, rainfall and temperature. [44] Typhoid and paratyphoid fever remain as an important public health problem at the global scale, and a major cause of morbidity in developing countries. [45] Typhoid symptoms include fever and abdominal pain, which without treatment can have a fatality rate of 10–30%. [46]

In Chile, there is a low incidence of typhoid and paratyphoid fever. [47] Even though they had high incidence and prevalence until the 90's, attributed to the consumption of contaminated drinking water and food. [48][49]

Additional to the links mentioned above between physicochemical environment that influence different stages of the transmission cycle, protected areas regulate diseases through the ecosystem changes associated with forest conservation that may filter pathogens from surface water, reduce flooding, maintain water quality and providing hydrological cycle stabilization. [11][19]

Moreover, biosphere changes caused by environmental destruction and fragmentation facilitate local emergence of zoonoses from their natural biotopes and their interaction with human population and domestic animals. [50]

Chile is facing a process of loss and decline of biodiversity and ecosystems. Half of its natural ecosystems show certain level of conservation threat related to changes in land-use, introduction and spread of invasive exotic species, development of primary production sector, forest fires and climate change. [51] Agents of many zoonoses have been detected in different wildlife environments, such as hydatidosis, salmonellosis and anthrax. [50]

Negative controls

Syphilis, Hepatitis B and Rubella were included as placebo outcomes. Syphilis is a highly contagious diseases spread mostly through sexual activity. It can cause abortions, neonatal death and in newborns disorders such as deafness, neurological deficit, growth retardation and bone deformities. In Chile, it is established that syphilis is a mandatory declaration infection and it must be notified daily to the Health Authority, by public and private healthcare establishments. [52] Hepatitis B is a viral and immune preventable disease and it is part of the National Immunization Program in Chile and it affects 0,15% of Chilean population. The only reservoir are humans and it can be transmitted parenterally, sexually, perinatally and horizontally through contact of mucosa. [53] [54] Rubella is a viral disease affecting susceptible children and young adults worldwide. It is transmitted in airborne droplets when people sneeze or cough. [55] During 1990, it was included in the National Immunization Program and from 1990 the incidence of rubella decreased rapidly in the country. [56]

Control variables. Regarding biophysical conditions, the climate of Chile encompasses a wide range of weather diversity throughout the country, depending on both latitude and altitude. In most of the country there are four seasons, with July and January being the coldest and warmest months respectively. Rainfall is more frequent during winter [20].

In terms of demographic factors, on average each municipality included in this study register on average 44,000 inhabitants. Most of them live in urban areas and are concentrated in central and southern areas due to climatic conditions and connectivity limitations. [57] Therefore distance to regional capital was also included, since it helps to control access to healthcare facilities and markets. [11]

b. Empirical Strategy

All analyses were carried out using Stata 16.1. Given the nature of the dependent variables (number of disease cases), we use a count data model. Whilst Poisson models (PRM) are the natural candidate to deal with count data, Negative Binomial Models (NBRM) were chosen given that our data proved to be over-dispersed (i.e. standard deviation higher than the conditional mean) and overly skewed to the right (i.e. high number of zeros). The appendix includes plots of predicted distributions calculated from PRM and NBRM. It can be easily seen that NBRM fits the distribution better for all of our dependent variables (se Figure A1).

To estimate the impact of protected areas on public health outcomes, we use a random effects model approach employing the variation of both between and within municipalities over time, and assuming that the unexplained residual variation is not related to independent variables. Since the relationship between diseases incidence and protected areas may vary at different levels of protection proportion, we estimated the marginal effects to show how these levels differ on impact on health outcomes.

Additionally, we included three negative control diseases whose incidence has no theoretical link with protected areas (Syphilis, Hepatitis B and Rubella) to support our empirical strategy and causal relationships. If our specification is correct, we should not find any impact between protected areas and the incidence of these diseases.

As it was mentioned before, the origin of protected areas is heterogeneous in time, and some of them are more than 100 years old. We do not ignore the effect that this might have on the impact on public health outcomes. Therefore, variables for protection period were included in 4 different categories: 0 to 20, 20 to 40, 40 to 60 and more than 60 years of protection.

Since there is no data for infectious diseases outcomes before 1999, when the SNASPE was established in 1984, we have no control over the outcomes baselines for assessing the impact of the conservation policy. Nevertheless, we empirically test that the independent variables used in our model also determine health outcome baseline data. To test this hypothesis, we compared the means for our health outcomes on a sample of municipalities that have never been protected with a matched sample of municipalities that started their protection after 1999. The municipalities were matched using Propensity Score Matching. To match these municipalities, variables of municipality land area, roads density, slope, altitude, average temperature in July and January, and rainfall were considered, resulting in 14 matched municipalities, 5 not protected at all (Sample 1) and 9 protected after the beginning of the studied period (Sample 2). Table 2 shows the difference in means between both groups. It can be seen, that there are not statistically significant differences between the average number of diagnosed cases in those municipalities that started their protection after 1999 and the 5 matched non protected municipalities for any disease.

Table 2

Mean comparison test between matched non protected municipalities and municipalities protected after 1999

	Mean Non-protected municipalities	<i>N</i>	Mean protected municipalities	<i>N</i>	Difference in means
(Para) Typhoid Fever	5.200	5	3.444	9	1.756
Echinococcosis	1.200	5	1.889	9	-0.689
Trichinosis	0.000	5	0.000	9	0.000
Anthrax	0.000	5	0.000	9	0.000
Syphilis	8.200	5	13.667	9	-5.467
Hepatitis B	0.800	5	1.111	9	-0.311
Rubella	7.000	5	5.444	9	1.556
<i>N</i>	14				
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$					

3. Results

a. Protected Areas

Table 3 presents the results from the random effects negative binomial model. Each column is labelled as the dependent variable for that specific model. As expected, our estimated parameter for protected areas are negative and significant for our outcome variables and not statistically significant for our negative controls. The results suggest that the highest the proportion of the municipality that is protected, the fewer the cases of each one of the diseases that were investigated.

Table 3
Negative Binomial Random Effects Regression Results

	(Para) Typhoid Fever	Echinococcosis	Trichinellosis	Anthrax	Syphilis	Hepatitis B	Rubella
Protected areas (% municipality area)	-1.482*	-3.018***	-8.109***	-10.746**	-0.590	-0.501	-0.574
	(0.886)	(0.979)	(1.919)	(4.227)	(0.623)	(0.998)	(1.116)
Number of inhabitants	0.000***	0.000***	0.000***	0.000	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Roads density (km/km ²)	0.126	-0.941***	0.722**	-3.584***	0.800***	0.588***	0.883***
	(0.144)	(0.209)	(0.319)	(1.036)	(0.104)	(0.160)	(0.183)
Distance to regional capital (km)	-0.001	0.004**	-0.003	-0.003	-0.001	-0.001	-0.005***
	(0.001)	(0.002)	(0.003)	(0.005)	(0.001)	(0.001)	(0.002)
Slope (°)	0.010	0.009	0.002	-0.041	0.010	0.002	0.040***
	(0.011)	(0.017)	(0.032)	(0.041)	(0.008)	(0.013)	(0.013)
Average altitude (m.a.s.l.)	-0.000*	-0.001***	-0.001**	-0.000	-0.000	-0.000***	0.000
	(0.000)	(0.000)	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)
Average temperature in July (°C)	-0.016	-0.203***	-0.078	-0.811***	0.018	0.052*	0.082***
	(0.028)	(0.039)	(0.073)	(0.142)	(0.020)	(0.030)	(0.031)
Average temperature in January (°C)	0.005	0.041	-0.004	0.594***	-0.059**	-0.146***	0.006
	(0.031)	(0.043)	(0.068)	(0.135)	(0.023)	(0.036)	(0.039)
Rainfall (ml)	-0.000	0.000	0.001***	0.001*	0.000	-0.001***	-0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
a ₀ to 20 years of protection	-0.246	0.119	0.905**	-0.986	-0.280**	-0.280	0.310

	(Para) Typhoid Fever	Echinococcosis	Trichinellosis	Anthrax	Syphilis	Hepatitis B	Rubella
	(0.185)	(0.254)	(0.355)	(1.079)	(0.123)	(0.196)	(0.230)
^a 20 to 40 years of protection	0.114	-0.259	0.273	-0.429	-0.341**	-0.397*	-0.584**
	(0.197)	(0.243)	(0.591)	(0.831)	(0.136)	(0.221)	(0.255)
^a 40 to 60 years of protection	-0.222	-0.023	0.826	-0.919	-0.144	-0.333	-0.133
	(0.233)	(0.285)	(0.512)	(0.846)	(0.164)	(0.286)	(0.291)
^a More than 60 years of protection	0.094	0.565**	1.565***	0.759	0.005	0.071	0.478*
	(0.186)	(0.227)	(0.294)	(0.554)	(0.136)	(0.238)	(0.256)
Year dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Constant	0.307	1.698**	-3.673***	-6.855***	0.963**	1.235*	-1.390*
	(0.595)	(0.851)	(1.315)	(2.126)	(0.480)	(0.745)	(0.798)
<i>N</i>	5,340	5,340	5,340	5,340	5,340	5,340	5,340
bic	11,286	10,136	2,474	627	23,228	9,654	6,216

b. Other covariates

Population density is positively correlated with each disease, but Anthrax. Municipalities with higher roads density have lower rates of Echinococcosis, Anthrax and all negative control diseases, but higher rates of Trichinosis. This might be explained by the adversarial effect of roads density, that improve both access to health services and damage ecosystems. [11] Altitude is negatively correlated with all diseases but Rubella. Average temperature in the coldest month (July) has a negative impact on Typhoid and Para Typhoid Fever, Echinococcosis, Trichinosis and Anthrax, but a positive effect on the negative control diseases Hepatitis B and Rubella. Average temperature in January has a positive effect on Anthrax and negative on Syphilis and Hepatitis B. Rainfall is positively correlated to Trichinosis and Anthrax and negatively correlated with Hepatitis B.

For the case of the number of years of protection, it is important to consider that although some of the protected areas are quite old, the creation of the national system of protected areas in 1984 represents the government effort to promote the definition and legalization of protected areas boundaries and the assignment of specific management objectives for each unit in the system, so it can be considered as the true beginning of protected areas in Chile. [58]

c. Marginal effects

In Fig. 1, we plot the marginal effects of the percentage of protected land area on the probability of finding a number of each disease higher than zero. It can be noticed that the marginal effects are always negative and decreasing in absolute terms.

For typhoid and paratyphoid fever and anthrax, the effect is not statistically significant at the lowest level of protection but becomes negatively significant when the protected area reaches around 20%. This indicates that there is a lower probability of finding cases of these diseases at higher levels of protection.

For trichinosis and echinococcosis, the effect is always negative and statistically significant. Slopes of marginal effects shown in Fig. 1 points that the effect is much higher at the starting levels of percentage of land being protected.

4. Conclusions

Anthropogenic activities cause ecosystem changes, but at the same time, humans are sensitive to changes in ecosystem services, affecting their own welfare [3]. Human health is included among the most underappreciated ecosystem service [59] and the particular relationship between health and ecosystem change has not been well documented [4][11]. At the same time, not all groups are similarly affected, since harmful effects of ecosystems degradation are being disproportionately borne by the poor. [1] The current global health crisis has shown the profound implications that a lack of an understanding between human health and ecosystem degradation can provoke.

Additionally, the lack of evidence about economic contributions of ecosystem services to people contributes to the perception that conservation initiatives, like protected areas, only constrain social benefits of economic extractive activities, thus involving only costs to local population. [11][19]

To help address this gap in knowledge and generating credible evidence for conservation practitioners and policymakers, we analyzed a rich dataset comprising human health outcomes and biophysical, land-use, demographic and socioeconomic factors. We found that protected areas decrease the incidence of infectious disease outcomes. Impact evaluation methodology produced robust estimates by showing no impact of protected areas on diseases not linked to ecosystem degradation. However, health dividends decrease with higher levels of protection which should be considered when designing future protection actions.

In summary, there are important health co-benefits that can be attributed to conservation policies. These important co-benefits should be taken into account when evaluating benefits and costs associated to future conservation decisions. This implies that the design of protected areas can be socially justified not only for their potential role on ecosystem preservation, but also for their important health benefits. By showing that protected areas decrease the incidence of various infectious diseases, the research provide evidence that conservation policies could provide more than just costs for local population and contribute to the wellbeing of the poorest and least entitled.

5. Declarations

a. Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

b. Ethics approval and consent to participate

Not applicable

c. Consent for publications

Not applicable

d. Competing interests

The authors declare that they have no competing interests

e. Funding

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f. Acknowledgments

Not applicable

References

1. Corvalán C, Hales S, McMichael AJ. Ecosystems and Human Well-Being: Health Synthesis. A Report of the Millennium Ecosystem Assessment. Washington, DC: Island Press; 2005.
2. Millennium Ecosystem Assessment. Ecosystems and Human Well-being: Synthesis. Press I, editor. Washington, DC; 2005.
3. Millennium Ecosystem Assessment. Ecosystems and Human Well-being: Biodiversity Synthesis. Washington, DC; 2005.
4. Myers SS, Gaffikin L, Golden CD, Ostfeld RS, Redford KH, Ricketts TH, et al. Human health impacts of ecosystem alteration. *Proc Natl Acad Sci U S A*. 2013;110:18753–60. doi:10.1073/pnas.1218656110.
5. Suzán G, Marcé E, Giermakowski JT, Mills JN, Ceballos G, Ostfeld RS, et al. Experimental Evidence for Reduced Rodent Diversity Causing Increased Hantavirus Prevalence. Wilby A, editor. *PLoS One*. 2009;4: e5461. doi:10.1371/journal.pone.0005461.
6. Colwell D, Dantas-Torres F, Otranto D. Vector-borne parasitic zoonoses: Emerging scenarios and new perspectives. *Vet Parasitol*. 2011;182:14–21.
7. Sutherst RW. Global change and human vulnerability to vector-borne diseases. *Clin Microbiol*. 2004;17:136–73.
8. Taylor D. Seeing the forests for more than the trees. *Env Heal Perspect*. 1997;105:1186–91.
9. Patz J, Daszak P, Tabor G, Aguirre A, Pearl M, Epstein J, et al. Unhealthy landscapes: Policy recommendations on land use change and infectious disease emergence. *Env Heal Perspect*. 2004;112:1092–8.
10. Diamond J. *Guns, germs and steel*. New York: W.W. Norton & Company, Inc.; 1997.

11. Bauch SC, Birkenbach AM, Pattanayak SK, Sills EO. Public health impacts of ecosystem change in the Brazilian Amazon. 2014. doi:10.1073/pnas.1406495111.
12. Corvalán AP. C. Preventing disease through healthy environments - Towards an estimate of the environmental burden of disease. editor. Geneva: WHO; 2006.
13. WWF. Equilibrium Research. Vital Sites. The contribution of protected areas to human health. 2010.
14. Disney LJ, Ruedas LA. Increased Host Species Diversity and Decreased Prevalence of Sin Nombre Virus. *Emerg Infect Dis.* 2009;15:7.
15. Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman J, et al. Global trends in emerging infectious diseases. *Nature.* 2008;451:990–4.
16. Taylor L, Latham S, Woolhouse M. Risk factors for human disease emergence. *Philos Trans R Soc London Series B Biol Sci.* 2001;356:983–9.
17. Lee PI, Hsueh PR. Emerging threats from zoonotic coronaviruses—from SARS and MERS to 2019-nCoV. *J Microbiol Immunol Infect.* 2020. doi:10.1016/j.jmii.2020.02.001.
18. Wilcox B, Ellis B. Los bosques y la aparición de nuevas enfermedades infecciosas en los seres humanos. *Unasylva.* 2006;57.
19. Pattanayak SK, Wendland KJ. Nature's care: diarrhea, watershed protection, and biodiversity conservation in Flores. *Indonesia Biodivers Conserv.* 2007;16:2801–19.
20. Instituto Nacional de Estadísticas. Compendio estadístico. Santiago de Chile; 2006.
21. OECD/ECLAC. Environmental Performance OECD, Reviews. Chile 2016. Paris: OECD Publishing; 2016. doi:https://doi.org/10.1787/19900090.
22. Pauchard A, Villarroel P. Protected Areas in Chile: History, Current Status and Challenges. *Nat Areas J.* 2002;22:318–30.
23. CONAF. Protección SNASPE.
24. Arriagada RA, Echeverría CM, Moya DE. Creating protected areas on public lands: Is there room for additional conservation? *PLoS One.* 2016;11. doi:10.1371/journal.pone.0148094.
25. Andam KS, Ferraro PJ, Pfaff A, Sanchez-Azofeifa GA, Robalino J a. Measuring the effectiveness of protected area networks in reducing deforestation. *Proc Natl Acad Sci U S A.* 2008;105: 16089–94. doi:10.1073/pnas.0800437105.
26. Pfaff A, Robalino J, Sanchez-Azofeifa GA, Andam KS. Park location affects forest protection: land characteristics cause differences in park impacts across Costa Rica. *B E J Econom Anal Policy.* 2009;9:1–26.
27. Wilson M. Infectious diseases: an ecological perspective. *Br Med J.* 1995;311:1681–4.
28. Myers SS, Patz J. Emerging Threats to Human Health from Global Environmental Change. *Annu Rev Environ Resour.* 2009;34:223–52.
29. Venegas J, Espinoza S, Sánchez G. Estimación del impacto económico de la equinococosis quística en Chile y análisis de las posibles causas que han dificultado su erradicación. *Rev méd Chile.* 2014;142.
30. Dakkak A. Echinococcosis/hydatidosis: A severe threat in Mediterranean countries. *Veterinary Parasitology.* Elsevier; 2010. pp. 2–11. doi:10.1016/j.vetpar.2010.08.009.
31. Martínez P. Hidatidosis humana: antecedentes generales y situación epidemiológica en Chile, 2001–2009. *Rev Chil infectología.* 2011;28:585–91.

32. Kazura JW. Tissue nematodes (trichinellosis, dracunculiasis, filariasis, loiasis, and onchocerciasis). In: eds. Bennett JE, Dolin R, Blaser MJ, eds Mandell. Douglas, and Bennett's Principles and Practice of Infectious Diseases, Updated Edition. 8th ed. Philadelphia: Elsevier Saunders; 2015. th. : .
33. Ribicich M, Gamble H, Bolpe J, Scialfa E, Krivokapich S, Cardillo N, et al. Trichinella infection in wild animals from endemic regions of Argentina. *Parasitol Res.* 2010;107:377–80.
34. Valencia C, Muñoz H, Torres M. Triquinosis. Entre el temor y el deber de informar la fuente de infección. *Rev Chil infectología.* 2003;20:99–103.
35. Hidalgo A, Oberg C, Fonseca-Salamanca F, Vidal M. Reporte del primer hallaz de puma infectado con *Trichinella* sp. en Chile. *Arch Med Vet.* 2013;45:203–6.
36. Murrell KD. The dynamics of *Trichinella spiralis* epidemiology: Out to pasture? *Vet Parasitol.* 2016;231:92–6. doi:10.1016/j.vetpar.2016.03.020.
37. Darwin Murrell K, Pozio E. Worldwide occurrence and impact of human trichinellosis, 1986–2009. *Emerg Infect Dis.* 2011;17:2194–202. doi:10.3201/eid1712.110896.
38. Hilmas CJ, Anderson J. Anthrax. *Handbook of Toxicology of Chemical Warfare Agents: Second Edition.* Elsevier Inc.; 2015. pp. 387–410. doi:10.1016/B978-0-12-800159-2.00029-4.
39. Shafazand S, Doyle R, Ruoss S, Weinacker A, Raffin TA. Inhalational anthrax: Epidemiology, diagnosis, and management. *Chest.* 1999;116:1369–76. doi:10.1378/chest.116.5.1369.
40. Perret C, Maggi L, Pavletic C, Vergara R, Abarca K, Dabanch J, et al. Ántrax (Carbunco). *Rev Chil infectología.* 2001;18.
41. Andreas S, editor. *Zoonoses - Infections Affecting Humans and Animals. Focus on Public Health Aspects.* Springer; 2015.
42. Sanz B. Las zoonosis transmisibles al hombre, en Chile. *Rev Univ (Universidad Católica Chile).* 1943;1:35.
43. Crump J, Mintz E. Global Trends in Typhoid and Paratyphoid Fever. *Clin Infect Dis.* 2010;50:241–6.
44. Dewan A, Corner R, Hashizume M, Ongee E. Typhoid Fever and Its Association with Environmental Factors in the Dhaka Metropolitan Area of Bangladesh: A Spatial and Time-Series Approach. *Plos Negl Trop Dis.* 2013;7.
45. John A, Crump SP, Luby. and EDM. The global burden of typhoid fever. *Bull World Health Organ Bull World Health Organ.* 2004;82:346–53. doi:10.1590/S0042-96862004000500008.
46. Buckle GC, Walker CLF, Black RE. Typhoid fever and paratyphoid fever: Systematic review to estimate global morbidity and mortality for 2010. *J Glob Health.* 2012;2:1–9. doi:10.7189/jogh.02.010401.
47. Ministerio de Salud de Chile. *Enfermedades transmitidas por alimentos. Informe de situación.* 2013.
48. Alvarez MDIL, Wurgaft F, Espinoza J, Araya M. Higiene habits and carriers in families with a child who has had typhoid fever * Hábitos de higiene e portadores em famílias que tiveram uma criança com febre tifóide. *Rev Saúde públ, S Paulo.* 1992;26: 75–81.
49. Cabello F, Agüero ME, Fernández ME. Epidemics of typhoid fever in Chile: ecological and microbiological aspects. *Rev méd Chile.* 1984;112:826–8.
50. Cabello CC, Cabello CF. Zoonosis con reservorios silvestres: Amenazas a la salud pública y a la economía. *Rev Med Chil.* 2008;136:385–93. doi:10.4067/s0034-98872008000300016.
51. UNDP. Policy Brief - Biodiversity in Chile: Suggestions to finance its conservation. BIOFIN Chile; 2017. p. 8.
52. Díaz J. Vigilancia epidemiológica de sífilis y gonorrea. *Rev Chil Infectol.* 2013;30:303–10. doi:10.4067/S0716-10182013000300005.

53. Chile M de S de. Situación Epidemiológica de la Hepatitis B y C. 2017.
54. Casanueva P. Vigilancia y monitoreo de las hepatitis virales B y C, exploración y análisis de datos. Santiago de Chile; 2015.
55. World Health Organisation. Rubella vaccines: WHO position paper-Recommendations. Wkly Epidemiol Rec. 2011. doi:10.1016/j.vaccine.2011.08.061.
56. Gallegos D, Olea A, Sotomayor V, González C, Muñoz JC, Ramos M, et al. Rubella outbreaks following virus importations: The experience of Chile. J Infect Dis. 2011;204. doi:10.1093/infdis/jir479.
57. Oficina de Estudios y Políticas Agrarias. Panorama de la Agricultura Chilena. Santiago de Chile; 2012.
58. Pauchard A, Villarroel P. Protected Areas in Chile: History, Current Status, and Challenges. Conserv Issues. 2002;22:318–30.
59. Pattanayak SK, Ross MT, Depro BM. CGE Evaluation of Health and Wealth Impacts Climate Change and Conservation in Brazil: CGE Evaluation of Health and Wealth. 2009;9.

Figures

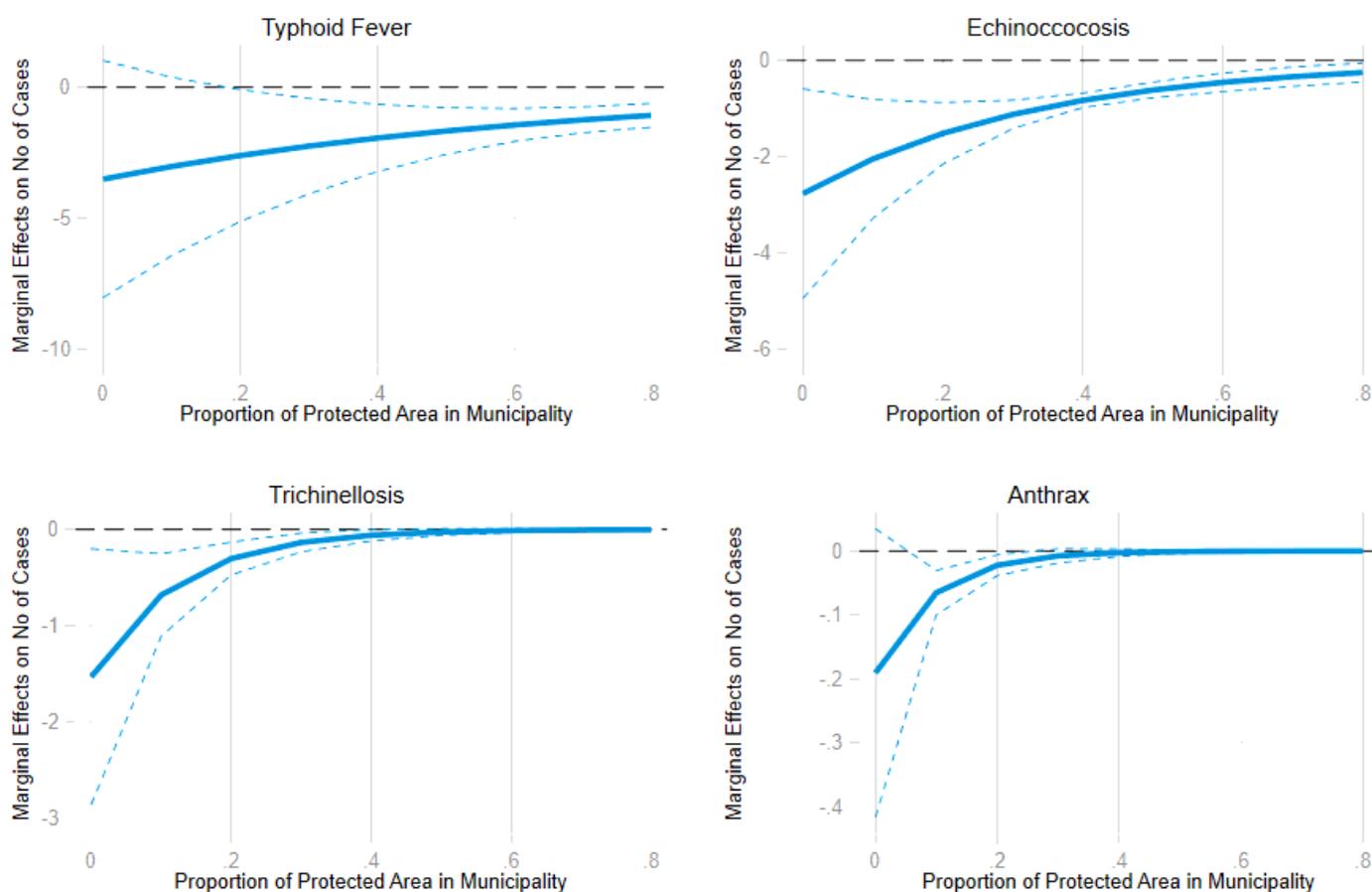


Figure 1

Marginal effects: The effect of a change on proportion of municipal protection on the number of disease cases

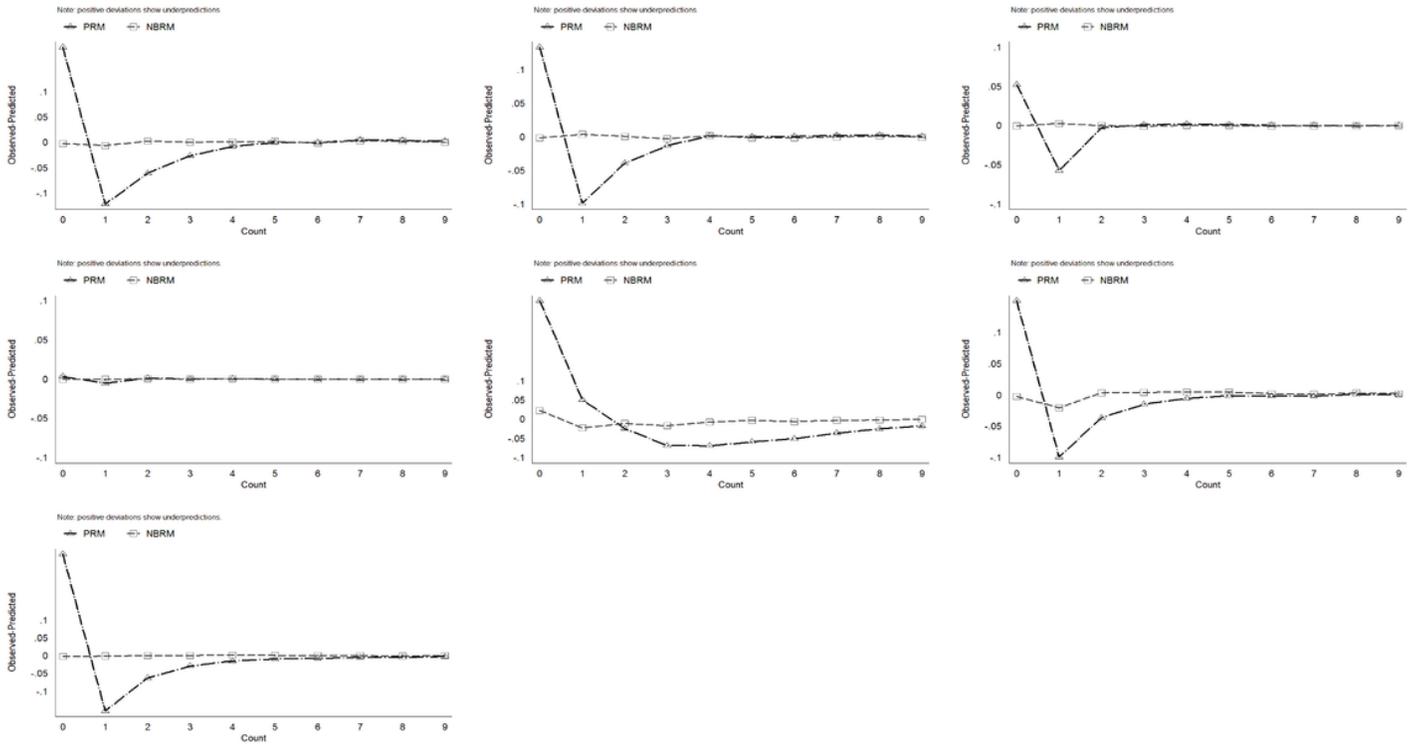


Figure 2

Model specification test. Poisson vs Negative Binomial.