Causal Mechanisms of Protected Areas on Poverty

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Abstract

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1 Introduction

In recent decades, protected areas have proliferated globally. These areas aim to restrict land uses and are often established on marginal lands in rural areas where poor households reside (Adams et al., 2004; Wilkie et al., 2006; Joppa and Pfaff, 2009; Sachs et al., 2009; Andam et al., 2010). These facts have led to increased efforts among scientists and practitioners to understand the economic effects of protected areas on nearby human populations (e.g., Robalino (2007); Robalino and Villalobos-Fiatt (2010)).

Few published studies, however, have the requisite data and methods to accurately estimate protected areas' causal effects on poverty (see reviews in Andam et al. (2010); Coad et al. (2008)). To properly account for changes in poverty due to the establishment of protected areas, a study should incorporate pre-protection, baseline measures of poverty (or proxies for them), and explicitly control for the non-random nature in which protected areas are established. The few studies that satisfy these criteria find that, contrary to conventional wisdom, the establishment of protected areas is associated with poverty reductions around protected areas in Bolivia, Costa Rica and Thailand (Canavire-Bacarezza and Hanauer, 2011; Andam et al., 2010; Sims, 2010). These studies, however, are unable to answer the question of *how* protected areas induce reductions in poverty.

From a policy standpoint understanding the mechanisms through which protected areas affect poverty is as important as identifying their average effects on poverty. In other words, we want to understand why, and in what context, protected areas can be expected to reduce poverty. Armed with such knowledge, decision-makers can design protected area networks to foster the mechanisms that alleviate poverty and to discourage the mechanisms that exacerbate poverty. The absence of credible estimates of causal mechanisms has been identified as a problem in all social policy fields, not just environmental policy (e.g., Deaton (2009); Heckman (2010)).

Unfortunately, identifying causal mechanisms is much more difficult than identifying average causal effects. First, one needs theory and field knowledge to help identify potential mechanisms. Then one needs measures of the mechanisms themselves (or proxies), rich data to control for characteristics of the lands and communities where protected areas are established that also affect the mechanisms and poverty outcomes, and an empirical design capable of identifying causal mechanism effects. Mechanisms are, by definition, affected by the causal factor of interest (e.g., protected

status) and thus simply controlling for mechanisms within, for example, a regression framework will generally lead to biased estimates (Rosenbaum, 1984).

To demonstrate how causal mechanisms of protected areas on poverty can be identified and quantified, we build on a rich data set from Costa Rica. These data were previously used to estimate the impact of Costa Rica's protected area system on poverty (Andam et al., 2010) and to characterize the way in which this impact varied conditional on observable characteristics of the local environment (Ferraro and Hanauer, 2011; Ferraro et al., 2011). We quantify the proportion of Andam et al. (2010) estimated poverty alleviation that arises from tourism, infrastructure development and ecosystem services mechanisms. We proxy for the respective mechanisms using the creation of park entrances, changes in road networks and changes in forest cover.

Another way to view our study is that it provides the first causal study, to our knowledge, of the poverty impacts of ecotourism at a national scale. The idea that nature-based tourism could benefit the rural poor has been vigorously debated (e.g,. Wunder (2001); Kiss (2004)), but little credible empirical evidence in favor or against the idea exists. Most studies or reviews are drawn from single-protected area case studies in which dubious assumptions about counterfactual poverty levels are implicitly invoked to permit causal inferences about protected area impacts.

Our analysis suggests that nearly half of the poverty reduction associated with the establishment of protected areas is causally attributable to tourism. Infrastructure development accounts for a relatively small proportion of the estimated poverty reduction. Ecosystem services from changes in forest cover account for no net effect on poverty. The remaining estimated poverty reduction comes from unidentified mechanisms, which may include mechanisms other than the three we identified or pathways not captured by our mechanism proxies (e.g., tourism opportunities unaffected by the presence or absence of a park entrance). We conduct several robustness checks that provide evidence that our inferences are not likely to be an artifact of our empirical strategy.

2 Data

In Andam et al. (2010), the unit of observation is the census tract. The 1973 census is used as the baseline year and demographic data are geocoded to their respective census tracts to form a set of covariates for each observation. In 1973 Costa Rica contained 4,694 census tracts with an average

size of 8.82km^2 (range: 0.00466-836 km^2). To determine if a census tract is considered protected for the analyses, a layer containing all protected areas established prior to 1980 is overlaid with the census tracts. As in Andam et al. (2010), a census tract is considered protected if at least 10% of its area is occupied by protected land.[1] Conversely, any census tract that contains less that 1% protected land is considered unprotected and a binary treatment indicator is assigned accordingly.[2] A poverty index is derived for each tract from census data following Cavatassi et al. (2004). Higher levels of poverty are associated with greater poverty index values (negative poverty index values indicate low levels of poverty). The censuses from which the poverty index is derived were conducted in 1973 and 2000. In the analyses, the poverty index calculation for 2000 is the outcome of interest. To match tracts on baseline characteristics, we use the matching covariates used in Andam et al. (2010), which include the 1973 poverty index and other baseline covariates that affect both protected area location and economic growth (see Table 1). The authors' calculation of average treatment effect on the treated (ATT) provides evidence that census tracts with protected areas that were established prior to 1980 had greater levels of poverty reduction between 1973 and 2000 than comparable unprotected census tracts.

2.1 Mechanisms

A causal mechanism can be viewed as a variable which, once affected by treatment, impacts the outcome of interest. In causal Directed Acyclic Graphs (DAGs) developed by Pearl (2009) and highlighted by Morgan and Winship (2007), a mechanism (S) is drawn as a causal pathway ($\rightarrow$) that links treatment (T) to outcome (Y), $T \rightarrow S \rightarrow Y$. Therefore, a causal mechanism is a variable whose quantity is directly affected by treatment, the result of which causes a direct change in the outcome of interest. A causal mechanism can thus also be viewed as an intermediate outcome variable in an elaborated theory of the causal relationship between T and Y.

[1] We use the 10% threshold in accordance with Andam et al. (2010). A 10% threshold was chosen because protecting 10% percent of the worlds' ecosystems was the goal of the 4th World Congress on National Parks and Protected Areas (Andam et al., 2010). Andam et al. (2010) show that their results are robust to changes in this threshold value (alternatively defined as 20% and 50%).

[2] Of the 4,691 census tracts, 249 are considered protected (treated) prior to 1980 and 4164 are considered potential counterfactual observations. To avoid bias in the analysis, 278 tracts with protection between one and ten percent are dropped from the analysis.

Table 1: Summary Statistics if Matching Covariates and Mechanism Variables

Covariate	Description	Mean	Median	Std. Dev.	Range
	Matching Covariates				
Baseline Poverty	Poverty index measured in 1973.	14.9	15.8	6.43	-6.4-28.9
Forest Cover	Percentage of census tract occupied by forest in 1960.	0.412	0.383	0.342	0-1
% High Productivity Land	Percent of census tract occupied by Land Use Capacity I, II or III land.	0.118	0	0.22	0-1
%Medium-High Productivity Land	Percent of census tract occupied by Land Use Capacity IV land.	0.295	0.04	0.377	0-1
%Medium-Low Productivity Land	Percent of census tract occupied by Land Use Capacity VI, VII or VIII land.	0.347	0.156	0.387	0-1
Distance to Major City	Average distance (km) from each 300m2 land plot within a census tract to nearest major city: Limon, Puntarenas or San Jose.	57.3	49.7	41.28	0.0037-208
Roadless Volume	The sum of the product of area and distance to nearest road (1969) for every 1 ha parcel within the census tract.	308,000	66,400	699,100	0.28-7,590,000
	Mechanism Variables				
Park Entrance	Binary indicator equal to 1 if census tract has at least 10% of its area occupied by a protected area with a park entrance	0.0276	0	0.164	0-1
Δ Roadless Volume	Change in roadless volume between 1969 and 1991	-8.76e+04	-1.75e+01	605191	-2.65e+07-6.32e+04
Δ Forest Cover	Percent change in forest cover between 1960 and 1986 within each census tract	0.0084	0	0.0926	0-0.75

2.1.1 Mechanisms in Costa Rica's Protected Area System

The putative mechanism through which protected areas achieve environmental outcomes is land-use restriction (e.g., preventing deforestation, logging, etc.). Such restrictions, which limit the exploitation of natural resources, would be expected to negatively impact economic conditions in surrounding areas. If land-use restrictions were the only mechanism (or the dominant mechanism) through which protected areas affect surrounding populations in Costa Rica, we would expect poverty to have been exacerbated, which has not been the case. Therefore, there must exist mechanisms through which protected areas have positively influenced economic conditions in surrounding populations. We consider three of the most plausible ones.

Tourism- Tourism is widely cited as a likely mechanism through which protected areas enhance local economies (e.g., Wilkie et al. (2006); Adams et al. (2004); Menkhaus and Lober (1996)). Costa Rica's stable government, rich biodiversity and protected area system make the country a popular destination for eco-tourists. Approximately 54% of international tourists to Costa Rica visit a protected area (ICT, 2010). Indirect evidence that tourism may play a role as a mechanism for poverty reduction comes from analyses of the heterogeneity of protected area impacts in Costa Rica. By measuring the way in which the poverty reduction impacts from protected areas vary with observable characteristics, Ferraro et al. (2011) provide indirect evidence for the reasons why protected areas succeed or fail to achieve environmental and social objectives. Most importantly for the purposes of our study, they find that reductions in poverty from the establishment of protected areas are greatest at intermediate distances to cities. This range coincides with the location of a majority of Costa Rica's national parks, which receive the most tourists. Further indirect evidence of tourism playing a causal mechanism comes from a recent study by Robalino and Villalobos-Fiatt (2010), which finds workers near park entrances in Costa Rica receive higher wages and are employed in higher-paid, non-agricultural activities than workers in similar areas farther from park entrances.

Using global positioning system (GPS) data from Robalino and Villalobos-Fiatt (2010), we proxy for the tourism mechanism by using observations of an establishment of a park entrance. Of Costa Rica's 39 protected areas that were established prior to 1980, 19 received at least one park entrance prior to 2000 (total of 23 entrances). A protected census tract (see definition above)

is considered exposed to a park entrance if it is occupied by a protected area in which at least one entrance was established. According to this assignment rule, 122 census tracts are considered exposed to a park entrance.

Infrastructure Development - Improved access and quality of infrastructure can be expected to enhance economic outcomes (e.g., reduce production costs). Previous studies from Costa Rica and Thailand have shown a relationship between access to urban infrastructure and poverty Andam et al. (2010). We proxy for the infrastructure mechanism with road networks. Access to roads increases access to markets and other resources (reducing transportation costs, etc.). In addition, roads serve as a good indicator of the level of infrastructure development and urbanization. We are therefore interested in how changes in road networks from the establishment of protected areas have impacted poverty in surrounding communities. *A priori*, the effect of protected areas on road networks is unclear: by restricting development, protected areas may reduce road network development (a negative mechanism) or, by encouraging law enforcement and tourism activities, they may increase road network development (a positive mechanism). We use changes in roadless volume (Watts et al., 2007) between 1969 and 1991 to capture the impact of changes in access to infrastructure. Roadless volume is an aggregation of the Euclidean distance to a road for each one-hectare land parcel within a census tract, adjusted for the size of the land parcel. Roadless volume is calculated by summing the product of the area of each land parcel (1 ha in this case) and the distance from that parcel to the nearest road (1969 and 1991). Therefore, higher measurements of roadless volume indicate fewer road networks within a municipality. Summary statistics for baseline roadless volume and changes in roadless volume can be found in Table 1.

Ecosystem Services - Since the seminal paper by Costanza et al. (1997), there has been great interest in quantifying the economic impacts of the services provided by intact ecosystems. One of the arguments from protected area advocates for potential 'win-win' outcomes is that the establishment of protected areas prevent ecosystem degradation (win) thereby providing a stream of economic benefits (win) to surrounding communities (in addition to the global benefits such as carbon sequestration). Given that the protected ecosystems are forest ecosystems, we proxy for ecosystem services mechanisms with the change in forest cover caused by protection (i.e., the difference in protected census tracts between observed forest cover and estimated counterfactual levels in the absence of protection). We are interested in how changes in forest cover from the establishment

of protected areas have impacted surrounding communities. We measure the percentage of forest cover within each census tract using GIS and forest cover boundaries from 1960 and 1986 (see Table 1 for baseline and mechanism measurements of forest cover). Note that, because we proxy for ecosystem services with changes in forest cover, which are also associated with potentially negative poverty mechanisms through foregone agricultural and other productive uses, we can only measure the net effect of the positive and negative channels through which this mechanism operates. For example, if the mechanism effects are positive, we can only say that ecosystem services affected by protection reduce poverty and this reduction is greater than any increase in poverty from forgone forest uses. If the mechanism effects are negative, we can only say that ecosystem services affected by protection have either no effect on poverty or their positive effect is smaller than the negative effects from forgone forest uses. If the mechanism effect is zero, either: (1) the effects on poverty from avoided ecosystem service losses and the opportunity costs of foregone productive uses are statistically indistinguishable from zero; or (2) the effects from avoided ecosystem service losses and of foregone agricultural uses are of opposite sign and approximately equal, thus cancelling each other out.

Clearly, our proxy variables do not capture all dimensions of our hypothesized mechanisms (e.g., infrastructure could be improved through improved road surfacing or through the provision of electricity, which would not be picked up in our mechanism variable). Moreover, there may be other mechanisms through which protected areas affect poverty in surrounding communities. Our methods, which are described in detail in the next section, do not require an exhaustive list of mechanisms and permit us to quantify the aggregate contribution of unidentified mechanisms.

3 Methods

3.1 Mechanism Concepts

To estimate the effect of protected areas on poverty, Andam et al. (2010) use a quasi-experimental design that assumes selection into treatment (protection) takes place on observable pre-treatment characteristics that may also affect poverty (i.e., confounding factors). They demonstrate how a failure to control for these characteristics substantially biases estimates of the treatment effect. In quasi-experimental designs, estimating the effect of a mechanism is confounded by the fact that

the mechanism is necessarily observed post-treatment and is potentially affected by treatment. Because a mechanism is affected by the treatment, controlling for it as if it were independent leads to biased estimates (Rosenbaum, 1984). Thus, one cannot simply estimate mechanism effects from the difference between the estimates of a specification (e.g., regression or matching) with and without the mechanism variable. The mechanism must be handled like an outcome variable (hence the concept of surrogate [outcome] variables in the epidemiology literature, see e.g., Mealli and Rubin (2003)).

3.2 Setup

To estimate the causal mechanisms through which protected areas have affected poverty, we use an augmented potential outcomes framework (we follow the framework and much of the notation of Flores and Flores-Lagunes (2011)). In the traditional potential outcome framework, there are two potential outcomes, $Y_i(1)$ and $Y_i(0)$, for each unit $i \in N$ (e.g., census tracts) under treatment ($T = 1$) and control conditions ($T = 0$). In other words, every unit has one outcome if it were exposed to the treatment and another outcome if treatment were withheld from it. The difference between these two potential outcomes, $\tau_i = Y_i(1) - Y_i(0)$, is the impact of the treatment on unit i. Unfortunately, for any given unit i only one of the two potential outcomes is observed. In practice, either unit i's outcome under treatment is observed given it was treated ($Y_i^{obs} \mid T = 1$) or unit i's outcome in the absence of treatment is observed given it was in the control condition ($Y_i^{obs}(0) \mid T = 0$). This inability to observe both potential outcomes is the fundamental problem for the estimation of causal effects. In order to calculate the average treatment effect in the absence of random assignment, it is necessary to invoke the conditional independence assumption (also known as ignorability, unconfoundedness or selection on observables), which states that potential outcomes are independent ($\perp\!\!\!\perp$) of treatment given a set of covariates X *that jointly determine outcomes and selection into treatment*:

Assumption 1 $Y_i(1), Y_i(0) \perp\!\!\!\perp T_i | X_i$,

Random assignment ensures independence, without condition, because each individual has an equal probability (or more generally, a probability known to the experimenter) of assignment to treatment. For conditional independence to hold under non-random assignment, one must condition

on (e.g., matching) or control for (e.g., regression) all covariates (X), thus rendering any remaining differences in outcomes between groups a function of treatment (or sampling error).

3.2.1 Principal Strata

Further complications arise when post-treatment mechanisms are introduced. Suppose S is a post-treatment mechanism that is measured at an intermediate period between administration of treatment and measurement of outcome.[3] Because, by definition, S is affected by treatment it is not unconditionally independent of treatment[4] and thus must be handled in a manner similar to the outcome of interest (Y). Therefore, as with Y, S has two potential outcomes $S_i(1)$ and $S_i(0)$ for each i, depending on assignment to treatment or control, respectively. This simply states that because mechanisms are affected by treatment, with the exception of some special cases, the mechanism outcome for each individual is dependent on the administered treatment. The implications, within the potential outcomes framework, are that four potential outcomes must now be considered for each individual: $(Y_i(1), Y_i(0), S_i(1), S_i(0))$.

There are now four compound potential outcomes of interest for i: $Y_i(1, S_i(1))$, the outcome when the unit is exposed to the treatment and the mechanism is affected by the treatment, which represents the total effect of treatment and is equivalent to $Y_i(1)$ in the traditional potential outcomes framework. $Y_i(1, S_i(0))$, the outcome when the unit is exposed to the treatment but the mechanism is not affected by the treatment (in other words, the outcome the unit would experience were we to expose it to the treatment but hold the value of the mechanism at its no-treatment value; in the language of Flores and Flores-Lagunes, the mechanism is "blocked"); $Y_i(0, S_i(0))$, the outcome when the unit is not exposed to the treatment and the mechanism is not affected, which is equivalent to $Y_i(0)$ in the traditional potential outcomes framework (i.e., post-treatment mechanism is not affected in absence of treatment); and $Y_i(0, S_i(1))$, the outcome when the unit is not exposed to the treatment but the mechanism is affected as it would be if the unit were treated. In general, only $Y_i(1, S_i(1))$ and $Y_i(0, S_i(0))$ are observed in practice, leaving $Y_i(1, S_i(0))$ and $Y_i(0, S_i(1))$ as counterfactuals that require estimation.

To help conceptualize the joint potential outcomes and identify the casual mechanism effect,

[3] Note that the three mechanisms of interest are denoted formally as S_j, where $j = 1, 2, 3$. For ease of exposition throughout a majority of this discussion, the subscript is omitted.

[4] This is true under random assignment of treatment as well.

we use the principal strata framework developed by Frangakis and Rubin (2002) (see also, Rubin (2004); Mealli and Rubin (2003)). Defining a principal stratum is similar to the concept of matching individuals (or groups of individuals) based on similar potential outcomes in a standard quasi-experimental setting. Two units from different treatments (e.g., protected, unprotected) share a principal stratum if they share potential mechanism outcomes (formally a principal stratum is defined where $\{S(0) = s_0, S(1) = s_1\}$.

To identify units from disparate treatments but similar principal strata, an extension to the conditional independence of Assumption 1 is necessary

Assumption 2 $S_i(1), S_i(0) \perp\!\!\!\perp T_i | X_i$.

In other words, we assume that, conditional on X, protection is not assigned based on expectations that tourism, infrastructure or ecosystem services will be different under treatment and control conditions. We call Assumption 2 conditional mechanism isolation. Assumption 2 states that potential mechanism outcomes are independent of treatment given a set of covariates (X) that *jointly determine selection into treatment and mechanism outcomes*, and, therefore, isolated from confounders. Under Assumption 2, we can identify units within similar principal strata: units from disparate treatments with similar values of X lie within common strata and, therefore, share similar potential mechanism outcomes. Assumptions 1 and 2 imply that potential outcomes and potential mechanism values are independent of treatment given covariates X. Combining Assumptions 1 and 2 we have

Assumption 3 $Y_i(1, S_i(1)), Y_i(1, S_i(0)), Y_i(0, S_i(0)), Y_i(0, S_i(1)), S_i(1), S_i(0) \perp\!\!\!\perp T_i | X_i$.

A necessary condition for satisfying Assumption 3 is that the covariates (X) must jointly determine selection into treatment, outcomes of interest and mechanism outcomes. Upon cursory examination, Assumption 3 may seem untenable. However, when one considers that the primary purpose of X is to control for the non-random process of selection into treatment and that treatment directly affects mechanisms, Assumption 3 seems more reasonable. Assumption 3 implies

$$E\left[S_i(1)|X_i = x, T = 1\right] = E\left[S_i(1)|X_i = x, T = 0\right] \quad (1)$$

$$E\left[S_i(0)|X_i = x, T = 1\right] = E\left[S_i(0)|X_i = x, T = 0\right]. \quad (2)$$

Equations (1) and (2) state that the expected mechanism outcomes under treatment, for units that were treated, are equal to the expected mechanism outcomes in the control group, for units with similar values of X, had they been treated, and *vice versa*.[5]

3.2.2 Estimands

In the study from which we draw (Andam et al., 2010), the estimand of interest is the average treatment effect on the treated, ATT: the effect of protection on poverty in the census tracts that were protected. Estimation of the ATT is akin to asking the question, "what would outcomes for protected census tracts have been had they not been treated?" Given that the total treatment effect estimand of interest is the ATT, the mechanism treatment effect of interest is the Mechanism Average Treatment Effect on the Treated ($MATT$). Thus, among the assumptions described in section 3.2, we only need make the following two assumptions

$$E[Y(0) \mid X, T = 1] = E[Y(0) \mid X, T = 0] \tag{3}$$

$$E[S(0) \mid X, T = 1] = E[S(0) \mid X, T = 0]. \tag{4}$$

Conditional on X, the expected poverty outcome and mechanism outcome in the absence of protection is the same for protected and unprotected units. We do not need the expected outcomes in the presence of protection to be the same.

Our estimands follow directly from the framework for mechanism average treatment effects ($MATE$) and net average treatment effects ($NATE$) developed by Flores and Flores and Flores-Lagunes (2011). A principal strata is defined as $\{S(0) = s_0,\ S(1) = s_1\}$, which states that individuals located within a common principal strata would have similar mechanism outcomes (s_0 had they been in the control group ($S(0)$), or s_1 had they been treated ($S(1)$), independent of actual treatment received). The $MATT$ can be written

[5]These are analogous to $E[Y(1) \mid X, T = 1] = E[(Y(1) \mid X, T = 0]$ and $E[Y(0) \mid X, T = 1] = E[(Y(0) \mid X, T = 0]$, which follow from Assumption 1. These equations are commonly used in the causal inference literature when selection is on observable characteristisc and demonstrate the equality (in expectation) of potential outcomes conditional on observable covariates (X) used to estimate average treatment effects.

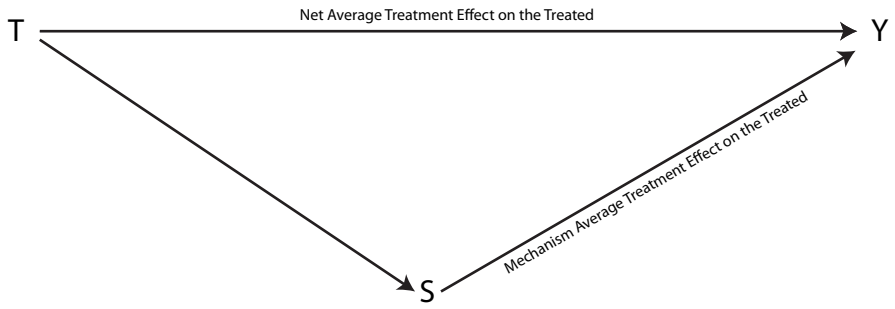


Figure 1: Causal Direct Acyclic Diagram (DAG) depicting the the concept of Mechanism Average Treatment Effect on the treated ($T \rightarrow S \rightarrow Y$) and Net Average Treatment Effect on the Treated ($T \rightarrow Y$) on outcome.

$$MATT = E\left\{E\left[Y_i(1, S_i(1)) - Y(1, S_i(0))|S_i(0) = s_0, S_i(1) = s_1, X_i = x, T = 1\right]\right\}. \tag{5}$$

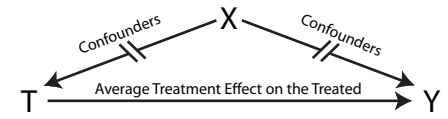
To estimate the $MATT$ one must ask, "what would outcomes for the treated have been, had they remained treated but treatment had not affected the mechanism?" Estimation of the $MATT$ answers this question by isolating the only source of variation in (5) to be the effect on outcomes due to a change in the mechanism (via blocking the effect of the mechanism on the outcome in the second term of (5)). A similar estimand of interest is the net average treatment effect on the treated ($NATT$) which isolates the effect on outcomes due to a change in treatment

$$NATT = E\left\{E\left[Y_i(1, S_i(0)) - Y_i(0, S_i(0))|S_i(0) = s_0, S_i(1) = s_1, X_i = x, T = 1\right]\right\}, \tag{6}$$

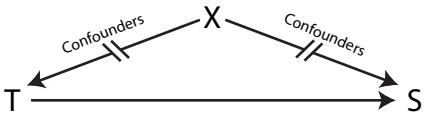
holding S at untreated levels. Estimation of $NATT$ is akin to asking, "what would outcomes for the treated have been, had they not been treated but their mechanism values remained at levels realized under treatment?" An advantage of the $MATT$ and $NATT$ is that they decompose the ATT such that $ATT = MATT + NATT$.[6,7] This decomposition states that the average treatment effect on the treated is equal to the proportion of the of treatment effect that is due to a change in the mechanism (induced by treatment), the $MATT$, and the proportion that is due to other

[6]Satisfaction of equation (4) is necessary for this identity to hold. Morgan and Winship (2007) outline conditions under which $T \rightarrow Y$ can be estimated using a set of mechanisms (e.g., the set of mechanisms is exhaustive and isolated). However, one can measure the partial effect of $T \rightarrow Y$ using a non-exhaustive set of mechanisms, S (i.e., $S \rightarrow Y$), which leads to an estimate of $MATT$. In conjunction with equation (4), under which unbiased estimates of the ATT can be estimated, the remaining difference between $MATT$ and ATT can be attributed to the mechanisms not included in S.

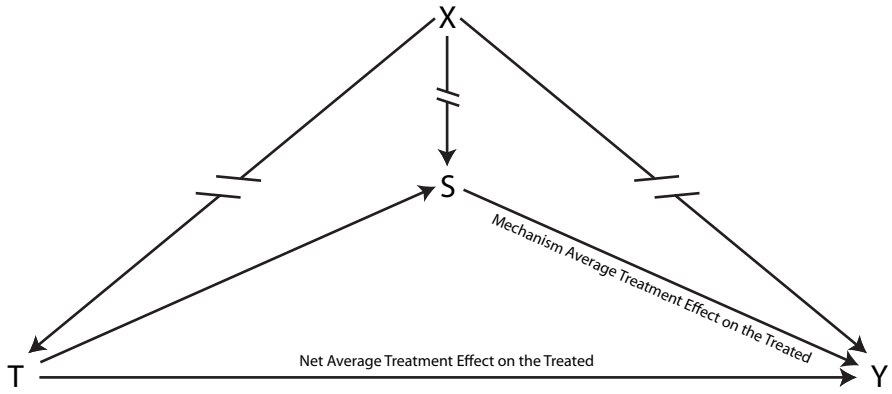
[7]The full decomposition can be written: $ATT = E[Y(1, S(1)) - Y(1, S(0))] + E[Y(1, S(0)) - Y(0, S(0))]$, given principal strata $\{S(0) = s_0, S(1) = s_1\}$.



(a) Assumption 1: Conditional Independence. Conditioning on covariates (X) that jointly determine selection inro treatment and outcome allows for unbiased estimation of the causal effect of T on Y.



(b) Assumption 2: Conditional Mechanism Isolation. Conditioning on covariates (X) that jointly determine selection inro treatment and post-treatment mechanism values allows identification of the causal effect of T on S and therefore principal strata.



(c) Assumption 3: Sequential Ignorability. Conditioning on covariates that jointly determine selection into treatment, outcomes of interest, *and* mechanism outcomes allows for estimation of Mechanism Average Treatment Effects on the Treated (MATT) and Net Average Treatment Effects on the Treated (NATT).

Figure 2: Directed Acyclic Graphs (DAGs) demonstrating the assumptions necessary for the causal estimation of ATT, $MATT$ and $NATT$. Each DAG shows how conditioning on observable covariates (X) breaks the confounding causal relationships ($T \leftarrow X \rightarrow Y$, $T \leftarrow X \rightarrow S$ and $S \leftarrow X \rightarrow Y$; represented by the broken single-headed arrows) and allows for estimation of ATT (a), the causal effect of treatment on mechanism outcomes (b) and $MATT$ (and $NATT$)(c).

mechanisms or solely to the effect of treatment, the $NATT$ (see Figure 1). Therefore, once either $MATT$ or $NATT$ is estimated the complementary estimate falls out of the difference with ATT.

3.3 Estimation Strategy

Estimation of either $MATT$ or $NATT$ is confounded by the fact that $Y_i(S_i(0))$ is typically unobservable.[8] We use matching in the first stage of the estimation to satisfy equations (3) and (4), see Figure 2. Post-matching, we follow methods suggested by Flores and Flores-Lagunes (2011). We use mechanism data from the matched control units, and a simple assumption about the way in which mechanisms affect outcomes within principal strata, to impute outcomes for treated units had treatment not affect the mechanism variables: the counterfactual of interest.

3.3.1 First Stage: Matching

We use one-to-one Mahalanobis covariate matching with replacement and post-match bias-adjustment (Abadie et al., 2004; Abadie and Imbens, 2006) to match control units to treated units. This approach serves two purposes. First, it provides an estimate of ATT , for comparison to $MATT$ and $NATT$, which offers comparability to previous studies from Costa Rica (Andam et al., 2010; Ferraro and Hanauer, 2011; Ferraro et al., 2011). Second, it provides a set of matched controls that, by Assumption 3 are within the same principal strata as the treated units to which they are matched. The latter purpose implies that the mechanism outcomes of the matched controls can be assumed to be the value observed by their treated counterparts, had treatment not affected the mechanisms. See Table 1 for a description of the covariates used for matching.

3.3.2 Second Stage: Estimate the Influence of Mechanisms

Flores and Flores-Lagunes (2011) suggest using a form of regression adjustment to impute outcomes for treated units had treatment not affected mechanisms. The necessary assumption for this approach (in addition to Assumption 3) is that the mechanism has a similar effect on potential outcomes $Y_i(1, S_i(1))$ and $Y_i(1, S_i(0))$, i.e., their conditional expectation functions share the same functional form (Flores and Flores-Lagunes, 2011).

[8] In the case where a subgroup of treated units for which treatment did not affect mechanism values can be identified, Flores and Flores-Lagunes (2011) develop an estimand for the local average treatment effect (LNATE), which requires less restrictive assumptions. See Section 5.3.2 for an application of this methodology to our data.

Assumption 4 *Suppose*

$$E\left[Y_i(1, S_i(1))|S_i(1), X_i = x, T = 1\right] = a_1 + b_1 S_i(1) + c_1 X, \tag{7}$$

then,

$$E\left[Y_i(1, S_i(0))|S_i(1), X_i = x, T = 1\right] = a_1 + b_1 S_i(0) + c_1 X_i. \tag{8}$$

Assumption 4 implies that the marginal effect of a change in the mechanism outcome has the same effect, on average, on units for whom exposure to treatment affects the mechanism as it does on units for whom exposure to treatment does not affect the mechanism.

In (7) and (8) of Assumption 4, b_1 represents the effect on outcome due to a change in the value of the mechanism S. The counterfactual of interest ($\widehat{Y}(1, \widehat{S}(0))$) can be estimated by evaluating (8), which uses the coefficients from (7), by setting $S_i(0) = E[S_i(0) \mid T = 1] = [\widehat{S}_i(0) \mid T = 1]$ which, according to 2, is equal to the observed control mechanism values within the common principal stratum of each treated unit.

Empirical estimation of the counterfactual of interest ($\widehat{Y}_i(1, \widehat{S}_i(0))$) is conducted by first running a regression of observed outcomes on covariate and mechanism values for treated units as in (7). Using the coefficients from this regression (a_1, b_1, c_1), we impute $\widehat{Y}_i(1, \widehat{S}_i(0))$ using the same treated unit covariates (as in (8)) and the matched control unit mechanism outcomes (where in (8) $S_i(0) = E[S_i(0) \mid T = 1] = S_i^{obs}(0)$ and $S_i^{obs}(0)$ is the observed mechanism outcome of each treated units respective matched control). Replacing the first term in (8), the empirical form for $MATT$ becomes

$$MATT = E\left\{E\left[Y_i^{obs}(1)|S_i^{obs}(1) = s_1, X_i = x, T = 1\right]\right\} - E\left[f_1(S_i(0), X_i)\right]. \tag{9}$$

Similarly, the empirical form of $NATT$ becomes

$$NATT = E\left[f_1(S_i(0), X_i)\right] - E\left\{E\left[Y_i^{obs}(0)|S_i^{obs}(0) = s_0, X_i = x, T = 1\right]\right\}, \tag{10}$$

where $f_i(S_i(0), X_i)$ in (9) and (10) is equal to $E\left[Y_i(1, S_i(0)) \mid S_i(1), X_i = x, T = 1\right]$ from (8).

We again emphasize the intuition behind the counterfactual of interest, which can be used in the estimation of both $MATT$ and $NATT$. The regression imputation methods presented in (7) and (8) allow us to address the question, "what would the outcomes for treated units have been

had their respective covariates $\left(X_i^{obs}|T=1\right)$ and influences of these covariates on outcomes (b_1) remained the same, but their mechanism taken on the values that would have been observed had they not been treated $(S(0)|T=1)$?" We note that the difference between $S_i^{obs}(1)|T=1$ (the observed mechanism value of treated units) and $\widehat{S}_i(0)|T=1$ (the estimated counterfactual values of treated units, had they not been treated) represents the unit-level causal effect of treatment on mechanism outcomes $(T \rightarrow S)$.

3.3.3 Bias-Adjusted Mechanism Outcomes

Abadie and Imbens (2006) and Abadie et al. (2004) suggest the use of post-match regression bias adjustments in the estimation of ATT to control for bias that remains from imperfect matching in finite samples. We apply a similar method in the estimation of our counterfactual mechanism outcomes.

Post-match bias-adjustment in estimation of ATT is conducted by first running a regression of outcomes on matching covariates $Y_{T=0} = X_{T=0}\beta_C + \epsilon$. This regression estimates the impact (β_C) of the matching covariates on outcomes for the matched control sample. To impute the ATT counterfactual of interest, β_C is combined with the covariates from the treated units $X_{T=1}$ to estimate $\widehat{Y}_{BA} = X_{T=1}\beta_C$: what treated unit outcomes would have been had their matching covariates had the same influence on outcomes as the control units. Note that if matching produces perfect balance across treated and matched control units then a counterfactual based on the observed values of the matched control outcomes $(Y_{i:T=0})$ will be identical to those estimated from the regression bias adjustment procedure $\left(\widehat{Y}_{i:BA}\right)$

The estimation of our counterfactual of interest in (9) is a function of both b_1 from (7) and $\widehat{S}_i(0)|T=1$. By Assumption 2, we can use the mechanism outcomes of the matched controls as the counterfactual for treated units. However, if imbalance in the baseline mechanism covariates remains after matching, we may be concerned that counterfactual mechanism values will be biased.[9] We, therefore, estimate our counterfactual mechanism values

$$\left[\widehat{S}_i(0)|T=1\right] = S_{i:T=0}^{obs} + \widehat{\mu}_0(X_{i:T=1}) - \widehat{\mu}_0(X_{i:T=0}) \tag{11}$$

[9]If mechanism outcomes are state dependent, then imbalance is a concern. For instance, if, after matching, unprotected tracts have lower baseline roadless volume, on average, than protected tracts, change in roadless volume may be less (in absolute terms) in unprotected tracts, simply because they started with larger road networks.

where $\widehat{\mu}_0$ represents the predicted values obtainedfrom combining the coefficients from a control group regression, of mechanism outcomes on covariates, with the respective treated ($\widehat{\mu}_0(X_{i:T=1})$) or control group ($\widehat{\mu}_0(X_{i:T=0})$) covariates. This procedure estimates the influence of baseline covariates on mechanism outcomes for control units and uses these estimated values to impute what the mechanism outcomes would have been had the control units been treated.

3.3.4 Standard Errors

To calculate the precision of our $MATT$ estimates we base our standard error estimator on the heteroskedasticity robust matching-based estimator suggested by Abadie and Imbens (2006).[10] Our estimator is calculated in two stages to allow for heteroskedastic variances within and across treatment arms. The variance for control units (for which comparison to $MATT$ is not meaningful) is calculated using a within treatment arm matching estimator. The Mahalanobis weighting matrix from the original matching process (used to create the matched sample) is used to find the nearest within treatment arm (unprotected) neighbor to estimate unit-level variances

$$\widehat{\sigma}^2_{i:T=0}(X_i) = (Y_i - Y_l)^2/2, \tag{12}$$

where Y_l represents the outcome of the nearest neighbor to unit i. The treatment level variance is then calculated

$$\widehat{V}_{T=0}\left(\widehat{MATT}\right) = \sum_{N_{T=0}} \lambda_i^2 \cdot \widehat{\sigma}^2_i(X_i), \tag{13}$$

where $\lambda_i = \#C_i/N_{T=0}$, and $\#C_i$ is the number oftimes that control unit i occurs in the set (was used as a match in the original matching specification).

The individual level variance for protected units is based on unit level deviations from the estimated $MATT$

$$\widehat{\sigma}^2_{i:T=1}(X_i) = \left(Y_i - \widehat{Y}_i(1, \widehat{S}(0)) - \widehat{MATT}\right)^2. \tag{14}$$

These unit level variances are then aggregated to calculate the treatment level (protected)

[10] A function that estimates the standard errors outlined in this section was programmed in R 2.11.1 and isavailable from the author upon request.

variance

$$\widehat{V}_{T=1}\left(\widehat{MATT}\right) = \frac{1}{N_{T=1}^2} \sum_{N_{T=1}} \widehat{\sigma}_{i:T=1}^2 \left(X_i\right). \tag{15}$$

The final $MATT$ standard error estimate is therefore

$$\widehat{\sigma}\left(\widehat{MATT}\right) = \sqrt{\left(\widehat{V}_{T=0} + \widehat{V}_{T=1}\right)}.$$

4 Results

4.1 Empirical Estimation of $MATT$

We conduct two distinct analyses to estimate the $MATT$ of our mechanisms of interest. First, the mechanisms are considered separately and the procedure outlined in the preceding sections is performed for each mechanism. Second, the mechanisms are considered jointly in the estimation of each $MATT$ via inclusion of all mechanisms in (7) and (8).

We begin by matching protected and unprotected census tracts using one-to-one Mahalanobis covariate matching with replacement. The resulting matched set (identical to the sample used by Andam et al. (2010)) comprises 249 protected and unprotected tracts, the covariate balance can be seen in Table 2. Using post-match regression bias-adjustment, the estimated ATT is -1.27, according to the poverty index. This result indicates that census tracts with at least 10% of their area occupied by a protected area prior to 1980 had differentially greater levels of poverty reduction (lower poverty index scores) between 1973 and 2000, on average, than comparable census tracts that remained unaffected by protected areas (see Andam et al. (2010) for full details).

4.1.1 Counterfactual Mechanism Values

The counterfactual of interest necessitates estimation of mechanism outcomes for treated units, had protection not affected the mechanism. For each mechanism, estimation of the counterfactual entails a two-step process. First, we estimate a matched unprotected group regression

$$S_{i:T=0} = X_{i:T=0}\beta_{1C} + \epsilon \tag{16}$$

Table 2: Balance Results for Matched Set. Mahalanobis one-to-one covariatematching with replacement.

Covariate	Status	Mean Prot.	Mean Unprot.	Diff. in Means	Norm. Diff.	Mean eQQ Diff.	%Improve MeanDiff.
Poverty Index	Unmatched	15.05	5.376	9.673	0.769	9.687	
1973	Matched	15.05	15.240	-0.187	0.013	1.64	98.07%
% Forest 1960	Unmatched	0.523	0.117	0.406	0.734	0.405	
	Matched	0.523	0.488	0.035	0.054	0.035	91.38%
% Land Use	Unmatched	0.093	0.304	-0.211	0.315	0.212	
Capacity 1,2,3	Matched	0.093	0.12	-0.028	0.060	0.03	86.84%
% Land Use	Unmatched	0.209	0.453	-0.244	0.330	0.245	
Capacity 4	Matched	0.209	0.20	0.009	0.016	0.026	96.19%
% Land Use	Unmatched	0.233	0.196	0.036	0.056	0.102	
Capacity 5,6,7	Matched	0.233	0.243	-0.011	0.019	0.034	70.98%
Distance to	Unmatched	58.53	34.87	23.670	0.286	23.62	
Major City	Matched	58.53	57.56	0.968	0.01	5.282	95.91%
Roadless	Unmatched	1113000	66830	1046000	0.321	1035000	
Volume 1969	Matched	1113000	681500	431600	0.110	440900	58.75%

where $S_{i:T=0}$ and $X_{i:T=0}$ represent the observed mechanism and baseline covariate values, respectively, of matched *unprotected* census tracts. The coefficients from (16) are then used to impute counterfactual mechanism outcomes for each mechanism

$$\left[\widehat{S}_i(0)|T=1\right] = X_{i:T=1}\widehat{\beta}_{1C} \tag{17}$$

where $X_{i:T=1}$ are the observed covariate values of the *protected* census tracts (empirical analog to equation (11))). Observed and counterfactual mechanism values for the protected census tracts can be seen in Table 3. The imputed counterfactual mechanism values from (16) are then used to calculate the counterfactual of interest: the outcomes for protected units, had protection not affected mechanisms ($\widehat{Y}_i(1, S_i(0))$).

Columns (i) and (ii) of Table 3 list the observed and estimated counterfactual mechanism values for the protected census tracts (see Table 4 for estimates of counterfactual mechanism values when bias-adjustment is not implemented). The counterfactual for our proxy for tourism is straight forward. Of the 122 census tracts that were impacted by a protected area with a park entrance, none would have a park entrance in the absence of protection. The estimated counterfactual for change in forest cover is significantly different from observed values as well. The average deforestation in protected census tracts between 1960 and 1986 was only 6.7%. We estimate that, had protection not affected deforestation, deforestation would have been approximately 23% (i.e., avoided deforestation

between 1960 and 1986 due to the establishment of protected areas was approximately 16.3%). Finally, we observe that there was greater infrastructure development (greater reduction in roadless volume) in protected census tracts between 1969 and 1991. However, the counterfactual measures of road networks are not significantly different from observed values.[11]

Table 3: Mechanism Results Using Mechanism Imputation

			Single Mechanism		All Mechanisms	
	(i) Baseline Mechanism	(ii) Counterfactual Mechanism	(iii) Mechanism Coefficient	(iv) $MATT$	(v) Mechanism Coefficient	(vi) $MATT$
Park Entrance	122	0	-1.004	-0.492 (0.439)	-1.345	-0.619 (0.448)
Δ Roadless Volume	-727,579	-674,147	2.694e-06	-0.143 (0.447)	2.790e-06	-0.148 (0.449)
Δ Forest Cover	-0.067	-0.23	0.627	0.103 (0.439)	0.124	0.02 (0.45)

(Heteroskedasticity robust standard errors)

4.1.2 Single Mechanism Estimation

In the single mechanism estimation the following procedure is run on each mechanism of interest independently. We first estimate the influence of covariates and mechanism on outcomes using the protected census tracts

$$Y_{i:T=1} = X_{i:T=1}\beta_{1T} + S_{i:T=1}\beta_{2T} + \epsilon \tag{18}$$

where $Y_{i:T=1}$, $X_{i:T=1}$ and $S_{i:T=1}$ are the observed outcomes, matching covariates and mechanism values for the protected census tracts,respectively. The counterfactual of interest is then estimated by obtaining the fitted values from

$$\widetilde{Y}_{i:T=1} = X_{i:T=1}\widehat{\beta}_{1T} + \widehat{S}_i\widehat{\beta}_{2T} \tag{19}$$

where $\widehat{S}_i = \left[\widehat{S}_i(0)|T=1\right]$ are the counterfactual mechanism values from (17), thus $\widetilde{Y}_{i:T=1} =$

[11] Differences are significant when counterfactual mechanism values are estimated without bias-adjustment. See Section 5.3.1 for results without mechanism imputation.

$\widehat{Y}_i(1, S_i(0))$. $MATT$ for each mechanism is calculated by subtracting the mean of the fitted values ($\widetilde{Y}_{i:T=1}$) from mean of the observed protected tract outcomes ($Y_i(1, S(1)) = Y_i(1)$).

Results from the single mechanism estimation strategy can be found in Columns (iii) and (iv) of Table 3. Column (iii) lists the estimated marginal impact of each mechanism ($\widehat{\beta}_{2T}$ from (19)) on poverty. Concordant with conjecture that protected areas have a positive impact on poverty by attracting tourism, we find that protected census tracts that were impacted by parks with entrances had lower poverty (by 1.04 according to the poverty index) than similar protected tracts. Because no protected tract would have been influenced by a park entrance in the absence of protection, the estimated $MATT$ (column (iv)) is -0.492. In other words, tourism, as measured by park entrances, accounted for approximately 40% of the poverty reduction associated with the establishment of protected areas.

The marginal impact of infrastructure development also has the expected sign (Column (iii)). Our results indicate that as road networks develop (roadless volume decreases) there is an associated reduction in poverty. We estimate that, had protection not affected road development in surrounding census tracts, there would have been less development in the absence of protection. However, the difference between observed and counterfactual values is relatively small. The slightly greater road development in protected census tracts accounts for a poverty reduction ($MATT$) of only -0.143 (approximately 11% of the total ATT).

The results for change in forest cover reflect the conflicting impacts underlying deforestation. There is a significant difference in observed and counterfactual deforestation in protected census tracts. We estimate that, had protection not affected deforestation, over 22% of the protected census tracts, on average, would have been deforested between 1960 and 1986 (compared to 6.7% observed deforestation). Despite this stark difference the $MATT$ of deforestation is quite small, 0.099, and indicates that the prevention of deforestation caused by the establishment of protected areas had essentially no impact on poverty.

4.1.3 Joint Mechanism Estimation

In the single mechanism estimation strategy each mechanism is considered independently. However, the estimated impact (according to $\widehat{\beta}_{2T}$) of a particular mechanism may be influenced by the inclusion or exclusion of additional mechanisms in (18). By including all of the mechanism variables

in (18) we allow the coefficients for each mechanism to reflect the presence of the other mechanisms. For clarity we denote the park entrance, change in roadless volume and deforestation mechanism variables as E, R and F respectively. The joint mechanism estimation analog to (18) is

$$Y_{i:T=1} = X_{i:T=1}\beta_{1T} + E_{i:T=1}\beta_{2T} + R_{i:T=1}\beta_{3T} + F_{i:T=1}\beta_{4T} + \epsilon \tag{20}$$

where all variables represent the observed values from the protected census tracts. The counterfactual of interest for each mechanism is estimated in a series of three imputations

$$\widetilde{Y}^{E}_{i:T=1} = X_{i:T=1}\widehat{\beta}_{1T} + \widehat{E}_i\widehat{\beta}_{2T} + R_i\widehat{\beta}_{3T} + F_i\widehat{\beta}_{4T} \tag{21}$$

$$\widetilde{Y}^{R}_{i:T=1} = X_{i:T=1}\widehat{\beta}_{1T} + E_i\widehat{\beta}_{2T} + \widehat{R}_i\widehat{\beta}_{3T} + F_i\widehat{\beta}_{4T} \tag{22}$$

$$\widetilde{Y}^{F}_{i:T=1} = X_{i:T=1}\widehat{\beta}_{1T} + E_i\widehat{\beta}_{2T} + R_i\widehat{\beta}_{3T} + \widehat{F}_i\widehat{\beta}_{4T} \tag{23}$$

where $\widehat{E_i}$, $\widehat{R}_i$ and $\widehat{F}_i$represent the imputed mechanism values from (17) (i.e., $\left[\widehat{S}_i(0) \mid T = 1\right]$ for the respective mechanisms). Equations (21) - (23) show that the counterfactual of interest for each mechanism is estimated by substituting the imputed mechanism values (from (17) for the mechanism of interest) into the respective equation, while leaving the covariates and complementary mechanism values at observed levels.[12] For instance, the counterfactual of interest for change in roadless volume ($\widetilde{Y}^{R}_{i:T=1}$) is calculated by plugging in the imputed counterfactual values for change in roadless volume ($\widehat{R}_i$) into the coefficients from (20), while leaving covariates ($X_{i:T=1}$) and mechanism values for park entrances (E_i) and change in forest cover (F_i) at the observed levels of protected census tract.

Results for the joint mechanism estimation strategy can be found in Columns (v) and (vi) of Table 3. We find that inclusion of all mechanisms in (18) does change the estimated influence of each mechanism (compare to Column (iii)): the coefficient on the park entrance mechanism increases in absolute terms from -1.004 to -1.345 (indicating a relative increase in poverty reduction attributable to tourism); the coefficient on the roadless volume mechanism increases from 2.694e-06 to 2.790e-06 (indicating a relative increase in poverty reduction attributable to infrastructure development), and

[12] A function that performs this iterative process was written in `R` 2.11.1 and is available from the author upon request.

the coefficient on the forest cover mechanism decreases from 0.627 to 0.124 (indicating a relative decrease in poverty exacerbation attributable to forest cover changes).

Under the joint mechanism estimation we find that the $MATT$ for the park entrance mechanism increases, in relative terms, to -0.619. This result implies that tourism associated with the establishment of protected areas accounts for approximately 49% of the estimated poverty reduction due to protection. Joint estimation also affects the $MATT$ for the deforestation mechanism which falls to 0.02. In other words, changes in forest cover from the establishment of protected areas have almost no impact of poverty. Joint mechanism estimation has a trivial effect on the $MATT$ of roadless volume which increases, in absolute terms, to -0.148.

4.2 Summary of Results

We estimate the $MATT$ for each of our mechanisms using both a single, and joint estimation strategy. Our results indicate that, while there are some differences, the choice of strategy is not driving the results or underlying implications. *A priori*, we prefer the joint estimation strategy because each mechanism coefficient (and, therefore, each $MATT$) accounts for the presence of the other mechanisms.

Of the mechanisms we consider, tourism accounts for greatest $MATT$, in absolute terms, and the greatest proportion of total poverty reduction due to the establishment of protected areas (estimated in the ATT). Nearly half of the poverty reduction associated with the establishment of protected areas is accounted for by our proxy for tourism, the establishment of a park entrance within a protected area. These results are concordant with anecdotal evidence, conjecture, and findings from a previous study (Robalino and Villalobos-Fiatt, 2010).

The development of infrastructure in protected census tracts has a strong poverty reducing influence as well (as measured by $\widehat{\beta}_{3T}$). However, because the establishment of protected areas did not substantially increase the road networks in the affected census tracts, compared to our counterfactual estimates, the $MATT$ on poverty was modest.

We find that change in forest cover associated with the establishment of protected areas (compared to counterfactual levels) has essentially no impact on poverty, as measured by the $MATT$. By measuring the impact of reductions in deforestation on poverty, due to protection, we were hoping to capture the impact of preserving ecosystem services. However, as mentioned in the In-

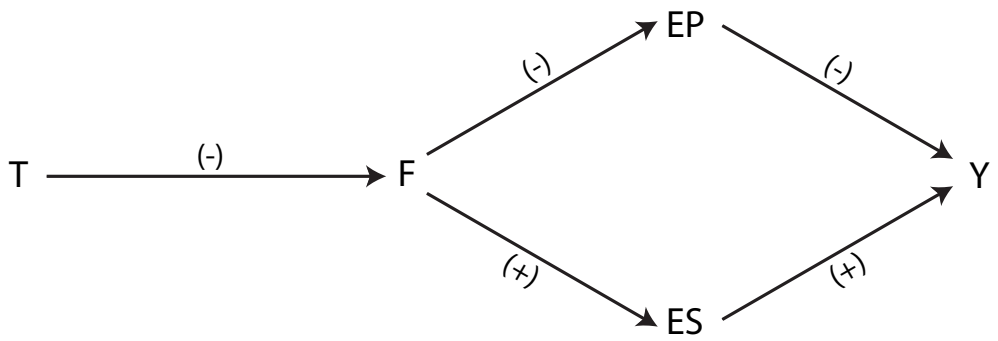


Figure 3: Direct Acyclic Diagram (DAG) depicting two of the potential underlying effects (and direction) of the change in forest cover mechanism. The the establishment of protected areas reduces deforestation $T \overset{(-)}{\rightarrow} F$. This causal reduction in deforestation has two impacts. First, we expect an increase in ecosystem services $F \overset{(+)}{\rightarrow} ES$ which would lead to a positive impact on poverty (poverty reduction), $ES \overset{(+)}{\rightarrow} Y$. Second, we expect the reduction in deforestation to decrease extraction profits $F \overset{(-)}{\rightarrow} EP$ which would lead to a negative impact on poverty (poverty exacerbation), $EP \overset{(-)}{\rightarrow} Y$. The relative magnitude of these countervailing effects determine the estimated $MATT$.

troduction, there are likely countervailing mechanism effects of avoided deforestation, which we believe are highlighted by our results. Figure 3 presents a DAG that depicts two of the potential underlying impacts that avoided deforestation would likely have on poverty. The establishment of protected areas reduces deforestation $T \overset{(-)}{\rightarrow} F$. This causal reduction in deforestation has two impacts. First, we expect an increase in ecosystem services $F \overset{(+)}{\rightarrow} ES$ which would lead to a positive impact on poverty (poverty reduction), $ES \overset{(+)}{\rightarrow} Y$. Second, we expect the reduction in deforestation to decrease extraction profits $F \overset{(-)}{\rightarrow} EP$ which would lead to a negative impact on poverty (poverty exacerbation), $EP \overset{(-)}{\rightarrow} Y$.[13]

Thus, without more data or assumptions, we cannot disentangle two competing explanations for our results: (1) the effects on poverty from avoided ecosystem service losses and the opportunity costs of foregone productive uses are both small or zero; or (2) the values of avoided ecosystem service losses and of foregone agricultural uses are of opposite sign and approximately equal, thus cancelling each other out. If one is willing to assert that restricting deforestation must have negative effects on poverty in surrounding communities, one could then infer that the protection of ecosystem services associated with the establishment of protected areas must provide countervailing poverty reducing impacts.

[13]This mechanism channel captures the concern the land-use restrictions associated with the establishment of protected areas may impose economic hardship by prohibiting extractive activities. Avoided deforestation provides an indication that land-use laws were binding and, therefore, is likely a valid channel.

4.3 Robustness

4.3.1 Without Mechanism Bias-Adjustment

In Section 3.3.3 we motivate and describe the use of bias-adjustment techniques to impute counterfactual mechanism values. We argue that, like the use of post-match regression bias-adjustments in the estimation of average treatment effects, this technique provides a (more) unbiased estimate of counterfactual mechanism values when imbalance persists post-matching (especially in baseline mechanism covari- ates). We re-estimate all $MATT$s without using bias-adjustment, the results can be found in Table 4. As expected, given the purpose of the bias-adjustment procedure, we find little difference in counterfactual values for change in forest cover (compare Column (ii) in Tables 3 and 4), for which a high degree of balance in baseline measures is achieved (see Table 2). In addition, because of the binary nature of the park entrance mechanism, the counterfactual values are identical with, and without, bias-adjustment. However, the counterfactual values for the roadless volume mechanism differ substantially. Without bias-adjustment the estimated counterfactual change in roadless volume is only -447,024 (compared to -674,147 with bias-adjustment). In turn, there is a much larger difference between observed and counterfactual roadless volume mechanism values which, thus, leads to much larger, in absolute terms, estimate of the $MATT$ (-0.7827). In other words, by not using bias-adjustment the estimated proportion of poverty reduction in the ATT attributable to protections causal effect on roadless volume, changes from approximately 11% to 61%. These results highlight that infrastructure development has a large influence on poverty reduction (as measured by $\widehat{\beta}_{3T}$). However, the magnitude of the associated $MATT$ is determined by the counterfactual mechanism value, which we believe is best estimated using the bias-adjustment framework that we outline in Section 3.3.3.

4.3.2 $LNATT$

The estimation of $MATT$ and $NATT$ requires the imputation of counterfactual mechanism values which are, by definition, unobserved. Our data provide a unique opportunity to estimate the causal effects of protection net of tourism under less restrictive assumptions than those used in the main analyses. We exploit the fact that some protected census tracts are observed in the absence of a park entrance mechanism. For this subset of the data $S_i(1) = S_i(0)$ by definition. In other words, the

Table 4: Mechanism Results Without Mechanism Imputation

			Single Mechanism		All Mechanisms	
	(i) Baseline Mechanism	(ii) Counterfactual Mechanism	(iii) Mechanism Coefficient	(iv) $MATT$	(v) Mechanism Coefficient	(vi) $MATT$
Park Entrance	122	0	-1.004	-0.492 (0.439)	-1.345	-0.619 (0.546)
Δ Roadless Volume	-727,579	-447,024	2.694e-06	-0.756 (0.54)	2.790e-06	-0.7827 (0.54)
Δ Forest Cover	-0.067	-0.223	0.627	0.099 (0.439)	0.124	0.019 (0.55)

(Heteroskedasticity robust standard errors)

potential park entrance mechanism value for protected units that did not receive an entrance is same under protection as it would have been in theabsence of protection ($S_i(1) = S_i(0) = s_0$). Therefore, we can identify this principal stratum ($\{S_i(1) = S_i(0) = s_0\}$) without invoking Assumption 2 or 3. In addition, we observe $Y_i(1, S_i(0))$ for this subset of the data and, therefore, do not need Assumption 4 to impute the counterfactual of interest.

The local $NATT$ ($LNATT$) can be estimated[14]

$$LNATT = E\{E[Y_i(1, S_i(0)) - Y_i(0, S_i(0))|S_i(1) = S_i(0) = s_0]\} \tag{24}$$

for the subset of data in the principal stratum$\{S_i(1) = S_i(0) = s_0\}$ (Flores and Flores-Lagunes, 2011). The fact that we observe protected census tracts that were not affected by a park entrance means that we can take the simple difference in these protected tract outcomes ($Y_i(1, S_i(0))$) and their matched controls ($Y_i(0, S_i(0))$), both of which are observed in the data. We estimate the $LNATT$ for this subgroup to be -0.6122. Flores and Flores-Lagunes (2011) note that the $LNATT$ represents the local ATT ($LATT$) for this subgroup because there is no mechanism effect for this group so $Y_i(1, (S_i(0)) = Y_i(1)$. Therefore, under Assumption 1, $LNATT = LATT = E[Y_i(1) - Y_i(0)|X_i]$ for this subgroup.

We note that the estimated $LNATT$ for park entrances is very close to the $NATT$ from the main analysis (-0.6122 and -0.659, respectively). We believe that the similarity between the two

[14]This framework follows directly from the framework for the local net average treatment effect ($LNATE$) established by Flores and Flores-Lagunes (2011).

estimates provides evidence that the assumptions and methods employed in the main analyses are providing unbiased estimates of the respective mechanism effects. We can make further comparisons to the $MATT$ estimates using the estimated $LNATT$ and an additional assumption of constant individual net effects (Flores and Flores-Lagunes, 2011)

Assumption 5 $Y_i(1, S_i(0)) - Y_i(0, S_i(0)) = C$, *for all i.*

Under this assumption we can define $LNATT = NATT$ and, therefore, estimate $MATT = ATT - LNATT$. Using this framework, the estimate of park entrance $MATT$ (-0.6658) is very close to the estimate from the main analysis (-0.619).

5 Discussion

One of the goals set forth by the 5^{th} World Parks Congress is that the establishment of protected areas should strive to reduce, and in no way exacerbate, poverty. To realize this goal, a greater understanding of the mechanisms through which protected areas affect poverty is needed. Recent impact evaluations from three developing countries have found that the establishment of protected areas has been associated with poverty reduction, on average, in surrounding communities (Canavire-Bacarezza and Hanauer, 2011; Andam et al., 2010; Sims, 2010). Although the average impact results from these studies are important, understanding the mechanisms through which protected areas affect poverty is arguably more important.

Using recently developed quasi-experimental methods, and rich biophysical and demographic data from Andam et al. (2010), we quantify the mechanism impacts of tourism, infrastructure development and ecosystem services on poverty due to the establishment of protected areas in Costa Rica prior to 1980. To capture the causal effects of these respective mechanisms, we use the establishment of park entrances and changes in road networks and forest cover as mechanism proxies. Our results indicate that approximately 50% of the poverty reduction estimated by Andam et al. (2010) can be attributed to tourism. Infrastructure development played a relatively small role in poverty reduction from protected areas. Finally, ecosystem services from impacts on forest cover accounted for no net effect on poverty, which implies either ecosystem services contribute nothing to poverty reduction or that their impact is equivalent to the damage done by protection's

restrictions on productive uses of the forest. The remaining estimated poverty reduction comes from unidentified mechanisms, which may include mechanisms other than the three we identified (e.g., social capital) or pathways not captured by our mechanism proxies (e.g., ecosystem services that do not depend on avoided deforestation). Future studies should seek richer measures of our mechanism variables. Richer mechanism variables will facilitate, for example, more precise measures of the impacts that enhanced supplies of ecosystem services from the establishment of protected areas have on poverty.

An understanding of both the heterogeneous impacts and mechanisms of protected areas might greatly improve the economic outcomes associated with the future establishment of protected areas. Nevertheless, the way in which protected areas affect surrounding populations will likely vary across countries. Costa Rica is a country renowned for its ecotourism investments and thus one should be cautious about extrapolating our results to protected area networks elsewhere. However, given the unresolved debate in the scientific and policy literature about the role of ecotourism in poverty alleviation, we believe our study highlights new avenues for research through the use of credible empirical designs aimed at identifying causal effects of ecotourism on poverty.

To truly understand the mechanisms through which environmental policies like protected areas affect poverty, we need to build the evidence base on a policy-by-policy and country-by-country (or region-by-region) basis. Ultimately, we need fully elaborated theories and structural empirical models through which we can fully understand the multiple causes of environmental and social outcomes and the tradeoffs among different policies. At this time, however, we are far from realizing this goal. Our best hope is to develop better theory and empirical evidence about the effects of individual causes (e.g., protected areas, incentive programs, decentralization) and slowly assemble a better understanding of the way in which our world operates.

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