

CHAPTER 2

USING THE PROPORTIONAL HAZARDS CURE MODEL TO IMPROVE THE STUDY OF INTERNATIONAL RELATIONS

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Abstract: Survival analysis has become an essential tool used by political scientists to study the timing and onset of diverse phenomena. However, scholars often use these models without regard for one of the fundamental assumptions they make, namely, that all observed subjects eventually experience the event of interest. Political scientists are often interested in events that could only feasibly occur among a subset of the subjects in their samples. Subjects that are not at risk of experiencing the event are often described as “cured” or “immune” to the event. Using standard models to analyze such data clearly violates the assumption above and may result in biased and inefficient coefficient estimates and lead scholars to make incorrect inferences. Cure models account for the presence of cured observations by modeling the probability of being at risk of experiencing an event of interest and reweighting the estimates of the hazard rate accordingly. This article makes three primary contributions. First, it introduces political scientists to the proportional hazards cure model (PHCM). Compared to the parametric cure models that have been used in political science thus far, the PHCM provides a flexible alternative that does not depend on restrictive distributional assumptions. Second, I present new software that I developed to estimate these models in R using time-varying covariates. Third, I demonstrate the potential advantages of using cure models by replicating an analysis of civil conflict recurrence.

2.1 Introduction

Survival analysis has become one of the fundamental components of the political scientist's methodological toolkit.¹ Scholars of international relations have applied these techniques widely to examine the duration and timing of diverse phenomena, such as regime change (Gates et al., 2016), cease-fire duration (Fortna, 2004), and war termination (Weisiger, 2013). Unfortunately, many scholars ignore one of the fundamental assumptions that standard survival models make: that all subjects will eventually experience the event of interest. Though this assumption is sometimes justified (e.g., all wars eventually end), it is often indefensible. A prime example is civil conflict recurrence; although some states may find themselves involved in civil wars again in the future, most will not (see, e.g., Walter, 2004).

The fact that some individuals are not at risk constitutes a form of unobserved heterogeneity and therefore has the potential to produce biased and inconsistent coefficient estimates. As a consequence, scholars cannot be confident that their results accurately approximate the substantive effects of their variables and cannot be certain that the results of significance tests are valid. To deal with these issues, scholars have developed models known as *cure models*. Developed primarily in the fields of biostatistics and medicine, cure models account for the fact that some subjects may be “cured” of a particular disease and are therefore “immune” to failure. Cure models typically account for this by assuming that subjects are drawn from two different populations: a group of subjects that are susceptible to experiencing the event of interest and another group of those who are not.²

The primary goal of this article is to introduce readers to cure models, and in particular, the semi-parametric proportional hazards cure model (PHCM). To date, cure models have rarely been used in the study of international relations and political science more generally (exceptions include Box-Steffensmeier, Radcliffe, and Bartels (2005), Clark and Nordstrom (2003), Findley and Teo (2006), Hettinger and Zorn

¹Survival analysis is also referred to by many different names including event-history analysis, duration analysis, and failure-time.

²I use the terms cured, immune, and unsusceptible synonymously throughout the manuscript to refer to subjects that are not at risk of experiencing an event. Likewise, I use uncured and susceptible interchangeably to refer to subjects that are at risk.

(2005), and Svolik (2008)). Each of these studies uses parametric cure models, which require making rigorous assumptions about the distribution of survival times. Incorrectly imposing a particular parametric form on the data represents a form of specification bias that potentially influences the results obtained (Box-Steffensmeier and Jones, 2004). Thus, in the absence of strong theoretical expectations about the shape of the baseline hazard, the PHCM provides a flexible alternative that is free of such assumptions.

The use of cure models also has theoretical advantages by allowing analysts to test whether a variable affects the probability that a subject is cured or affects the hazard rate conditional on the fact that a subject is susceptible. Which process a variable influences alters the interpretation of that variable and may therefore provide more nuanced understandings of how they affect the dependent variable. For example, when studying the duration of peace after conflict, it is of interest to know whether a variable decreases susceptibility to conflict onset or merely increases the time until conflict recurrence. Standard duration models can only tell us that a variable extends the duration of peace, but cannot distinguish between these mechanisms.

I also present a new R package I developed to estimate the PHCM in R using time-varying covariates. Existing software packages are incapable of incorporating data using time-varying covariates and therefore only allow scholars to analyze purely cross-sectional data. This represents a substantial limitation on the types of phenomena and data that can be used. As such, releasing this open-source software package will greatly expand the ability of analysts to apply these models in a variety of fields. To compare the performance of the standard Cox model and PHCM, I replicate an analysis of civil conflict recurrence by Loyle and Appel (2017). The results demonstrate that the two models produce different inferences and lead to large differences in the size of the substantive results.

The rest of this chapter proceeds as follows. Section 2.2 discusses the problems associated with the presence or cured observations. It demonstrates that cured observations have the potential to produce biased and inconsistent estimates of the regression parameters and discusses how analysts can determine whether a sample contains cured observations. Section 2.3 introduces the PHCM, discusses the relative advantages and disadvantages of using parametric and semiparametric cure models, and discusses how to

test the proportional hazards assumption. Section 2.4 describes the software I developed to implement the PHCM in R. Section 2.5 discusses the advantages that cure models have over standard survival models when it comes to theory-testing and outlines how cure models can be used to improve tests of theories of international relations. Section 2.6 presents the replication analysis of civil conflict recurrence. Section 2.7 concludes.

2.2 The Problem of Cured Observations

The fundamental problem raised by the presence of cured individuals is the fact that they have the potential to introduce bias, which may in turn lead to incorrect substantive effects and inferences. To illustrate why, let T be a positive, random variable representing the time at which subjects experience the event of interest, referred to as the *survival time* or *failure time*. The survival function, $S(t)$, describes the probability that a subject survives at least until time t , given by $S(t) = \Pr(T \geq t)$. Subjects that are not observed to fail by the end of the observation period are considered *right-censored*. Whether a subject's failure is observed is recorded by a dummy variable, δ , equal to one if a subject fails and zero if not.

Standard survival models make the implicit assumption that all subjects eventually fail, i.e., that $S(t)$ approaches zero as t goes to infinity. Although some observations may be right-censored, this fact is typically attributed to the fact that some subjects simply have failure times that exceed the time span covered by the available data. However, the prospect of cured individuals provides an alternative reason why subjects may be right-censored. It is useful to conceive of a population of subjects containing cured individuals as being drawn from two different subpopulations: one that is susceptible to failure and another that is not.

Systematic differences in the two subpopulations constitute unobserved heterogeneity and therefore manifest as endogeneity. This leads the model to produce estimates of the survival function and covariate effects that are biased and inconsistent. Cured observations are analogous to the “excess zeroes” that may arise when working with count data and ordinal dependent variables, among others (e.g., Bagozzi et al., 2015; Lambert, 1992). Many scholars of international relations are familiar with the problems of

including observations that are not at risk in other contexts, such as when data contain excess zeroes or when dealing with rare events. For example, count data may contain subjects that cannot experience an event for some reason (“structural” or “excess” zeroes) and those that simply did not experience an event (“random zeroes”). If the excess and random zeroes differ systematically, the use of standard event count models will produce bias. Likewise, in the context of rare events, scholars often attempt to eliminate subjects that are not at risk by removing certain observations from the sample. For example, scholars often attempt to limit the sample to the subjects that are most at risk of experiencing an event by only including politically relevant dyads. Another approach taken by Xiang (2010) is to model this unobserved heterogeneity using split population binary response models (see below).

In practice, including cured subjects increases the number of subjects with long survival times. As a result, conventional models will overestimate the probability of survival among the susceptible population and, conversely, underestimate the hazard rate. Likewise, the model will overpredict the survival time of individuals that are susceptible and underpredict the (infinite) survival time of those who are not (Beger et al., 2017). Since the unobserved differences across populations introduce heterogeneity in covariates’ effects, these models will also produce biased and inconsistent estimates of the coefficients. Coefficients may be either too small or too large depending on the nature of the differences between the two populations.

Ideally, the easiest way to deal with cured subjects would be to simply remove them from the sample to obtain unbiased estimates of the hazard rate among the uncured subjects. The difficulty of doing this arises from the fact that they typically cannot be identified a priori. Although the status of the subjects whose failure is observed is known, those that are right-censored may belong to either class. The presence of a large number of right-censored observations is not necessarily an indicator that a cure model is necessary. Heavy censoring may merely be an indicator that a study was not run long enough to observe the failure of most of the subjects.

Determining whether the right-censored observations in a sample contain cured subjects is primarily a theoretical exercise. Sy and Taylor (2000) state that “there are a number of ways that one might address

whether a cure model is appropriate, the most important of which is having a biological rationale from the underlying science,” (234). Translated to the realm of social science, analysts must consider whether the assumption that all subjects fail makes theoretical sense in terms of the phenomenon they are evaluating. In cases where the assumption that all units fail is plausible, the presence of a large number of right-censored observations is likely due to inadequate follow-up time. However, when there is strong reason to believe that some subjects will not fail, standard duration models will not accurately reflect the data-generating process that produced the observed data. The second condition is more likely to hold when examining conflict data, for example, since there are often conflict-free periods that span decades (see the replication analysis below).

Empirically, the extent to which long-term survivors are present in the data can be assessed using a nonparametric Kaplan-Meier survival curve. If the Kaplan-Meier estimates approach zero, it provides evidence that the right-censored subjects are likely to eventually fail. However, the presence of a long right tail that plateaus above zero is a likely indicator that there is a set of individuals in the population that survive long after most susceptible subjects have failed Sy and Taylor (2000).

2.3 The Proportional Hazards Cure Model

Cure models deal with the fact that the cure status of right-censored subjects is unknown by assuming that the cure process is probabilistic. Most cure models, take the form of *mixture models*, also known as *split-population models*.³ Mixture models are a broad class of models used when a population is composed of multiple latent subpopulations that cannot be fully separated a priori. They assign each subject a probability of belonging to a particular class and then weight their contribution to the likelihood function accordingly. Many political scientists are already familiar with mixture models in the form of zero-inflated models, which assume that some subset of the observations cannot take on values above zero (Lambert, 1992; Bagozzi et al., 2015).

³An alternative class of models known as *nonmixture* cure models corrects for these problems by imposing an upper bound on the cumulative hazard rate. Although these models have been used widely in biostatistics, they have seen limited applications in social scientific fields and are not considered further here.

Specifying a mixture cure model entails modeling the population survival function as a function of both the probability of failure and the survival function for those observations that do fail. Let p represent the probability of belonging to the susceptible class and $1 - p$ represent the probability of being cured. Conditional on being in the uncured class, the probability of surviving until at least time t is given by $S_u(t)$, referred to as the *conditional survival function*. For individuals in the cured class, the survival function can be assumed to equal one at all t . The probability that an uncured individual is alive at time t is thus $1 \times (1 - p)$, which reduces to $1 - p$. The overall probability of being alive at time t for an individual randomly drawn from the population is thus given by

$$S_{pop}(t) = pS_u(t) + (1 - p), \quad (2.1)$$

where S_{pop} is referred to as the *population survival function* or *marginal survival function*.

Covariates can be incorporated into Equation 2.1 by constructing models for p and $S_u(t)$. The proportional hazards cure model (PHCM) is a variant of mixture cure model that uses the standard Cox model survival function to model $S_u(t)$. If \mathbf{x} is a vector of covariates and $\boldsymbol{\beta}$ is a vector of associated regression coefficients, the formula for the conditional survival function is given by

$$S_u(t) = S_{u0}(t)^{\exp(\mathbf{x}\boldsymbol{\beta}')} , \quad (2.2)$$

where $S_{u0}(t)$ is the *baseline conditional survival function* that describes how susceptible subjects' probability of failure changes over time, independent of covariates. Substituting Equation 2.2 into Equation 2.1 yields the semiparametric mixture cure model:

$$S_{pop}(t) = p[S_{u0}(t)^{\exp(x\boldsymbol{\beta})}] + (1 - p). \quad (2.3)$$

The coefficients in Equation 2.3 are interpreted in the same way as the standard Cox model. Positive coefficients indicate that a variable is positively correlated with the hazard rate, and therefore associated

with decreased survival times. As with the standard Cox model, the PHCM requires assuming that the effect of covariates is proportional over time (discussed further below).

Whether a subject is susceptible to an event be modeled as a function of covariates using a binary-response model to construct a model of p . Let Y be a dependent variable coded one if a subject eventually fails and zero otherwise. Further, let \mathbf{z} be a vector of covariates and their associated coefficients be represented by γ . The probability that a subject is at risk for failure is typically modeled using a logistic regression model, given by

$$p = \Pr(Y = 1) = \frac{\exp(\mathbf{z}\gamma')}{1 + \exp(\mathbf{z}\gamma')}. \quad (2.4)$$

The use of other binary response models such as a probit model is also possible. Analysts can also allow covariates to have a non-linear additive effect on the probability of failure by using generalized additive models in place of a binomial generalized linear model for γ (Peng, 2003; Ramires et al., 2018).

Determining which covariates to include in each equation is a theoretical exercise that depends on the causal mechanism connecting a covariate to the observed survival times. Variables that are thought to increase or decrease an individual's susceptibility should be included in the cure equation, while those thought to shorten or extend the time until a susceptible subject experiences an event should be included in the hazard equation.

It is important to note that there is no restriction on including variables in both the cure and hazard equations. This allows for the possibility that a variable may influence both subjects' susceptibility at large and the timing of failure among the class of susceptible individuals. Incorporating a variable in both equations makes it possible to test whether an independent variable influences p , S_u , or both. For example, it is possible to test whether a treatment increases the probability that an individual survives long-term or merely extends the time until susceptible patients die. Likewise, peacekeeping operations may be thought to eliminate the possibility of a civil war while also decreasing the hazard rate among susceptible observations.

The PHCM can easily accommodate time-varying covariates using the “counting process” or “start-stop” data structure to include one observation for each subject in the riskset at each of the observed failure times (see, e.g., Box-Steffensmeier and Jones, 2004, Chapter 7). It is important to note, however, that incorporating time-varying covariates changes the interpretation of the cure portion of the model. Rather than merely identifying which individuals are and are not susceptible, the inclusion of time-varying covariates introduces the possibility that subjects may be susceptible at some times and not at others. Although this can make substantive sense, it complicates the interpretation of the variables in the cure equation. It may thus be easier to include only time-invariant or slowly-changing covariates in the cure equation (Beger et al., 2017; Dirick et al., 2017).⁴

Estimating the parameters for the PHCM can be estimated using maximum likelihood methods. The complete data log-likelihood for Equation 2.3 is given by

$$\mathcal{L}_C(\Theta) = \prod_{i=1}^n p_i^{y_i} (1 - p_i)^{1-y_i} \prod_{i=1}^n \{h_u(t_i)^{\delta_i y_i} (S_u(t_i))^{y_i}\}. \quad (2.5)$$

The first product term in Equation 2.5 contains the parameters related to the cure component of the model, where p_i is the probability that a subject is susceptible to the event and y_i is a binary indicator of whether a subject eventually fails. For subjects that eventually fail, this term reduces to p_i , while for those that do not, it reduces to $(1-p_i)$.

The second product term in Equation 2.5 contains the parameters related to the hazard component, where $h_u(t_i)$ represents the baseline hazard rate for subjects that are not cured and δ_i is a censoring indicator coded one if a subject is observed to fail and zero if not. For subjects that are not cured (i.e., $y_i = 1$), this term reduces to

$$h_u(t_i)^{\delta_i} S_u(t_i). \quad (2.6)$$

⁴This does not imply that time-invariant covariates must be included in the cure equation or that they should not be included in the hazard equation.

Subjects that are observed to fail (i.e., $\delta_i = 1$) contribute information to both the hazard term and survival term in Equation 2.6. However, for subjects that are not observed to fail (i.e., $\delta_i = 0$), the hazard term reduces to one. This is because subjects that are not observed to fail do not contribute information about the hazard function or failure times. For subjects that are cured (i.e., $y_i = 0$), the product term reduces to a value of one as each term is raised to the zeroth power. This is because cured subjects do not contribute information about the failure time and survival time of uncured subjects.

Maximizing the likelihood function in Equation 2.5 is difficult because y_i is not known for all subjects. Although $y_i = 1$ for subjects that are observed to fail, it is unknown whether censored subjects are cured or not. Put otherwise, for subjects that are not observed to fail, y_i could equal zero or one. To deal with this, Peng and Dear (2000) and Sy and Taylor (2000) derived an expectation maximization algorithm to obtain the maximum likelihood estimates by iteratively estimating the model parameters (β and γ) and using these to estimate the value of y_i . The algorithm and estimation procedure is described in full in Appendix A.1.

Compared to parametric cure models, the PHCM has two advantages. First the validity of the results obtained from parametric models depends upon choosing an appropriate distribution for the failure times. Using a distribution that does not accurately describe the baseline survival function constitutes specification bias and thereby produces biased and inconsistent estimates of the model's parameters. In many cases it is difficult to verify that a particular parametric distribution is appropriate. As such, without strong theory or evidence to support the use of a particular distribution, semiparametric approaches that leave the baseline hazard unspecified are a safer alternative.

Second, simulation studies demonstrate that the PHCM produces more efficient estimates of β and γ than parametric cure models under certain conditions (Kuk and Chen, 1992; Sy and Taylor, 2000). When censoring is mild and a parametric model accurately describes the baseline survival function, the parametric cure model tends to be more efficient. This is comparable to standard survival models, where parametric models are more efficient when they correctly specify the baseline hazard (see Box-Steffensmeier and Jones, 2004). However, when there are high levels of censoring, the PHCM tends to be more efficient,

even when the parametric model accurately describes the baseline survival function. This is due to the fact that the procedure used to estimate semiparametric cure models constrains S_{u0} to be zero following the final failure time (see Appendix A.1 for more details). As a result, the PHCM has an advantage when dealing with rare events data.

On the other hand, the PHCM depends on the proportional hazards assumption. Violations of the proportional hazards assumption create the potential for biased coefficient estimates and standard errors (Box-Steffensmeier and Zorn, 2001). The proportional hazards assumption also has implications for the interpretation of coefficients and the substantive conclusions drawn from a model. The effect of many social scientific variables may be expected to change over time. As Box-Steffensmeier and Zorn (2001) note, “the influence of an independent variable may be greater or smaller, or even change signs, depending on the amount of time that has elapsed for that observation,” (974).

As such, it is necessary to test whether each covariate violates the proportional hazards assumption. One common method of detecting violations of the proportional hazards assumption in standard survival models is by assessing the Schoenfeld residuals (Grambsch and Therneau, 1994; Schoenfeld, 1982). Schoenfeld residuals are covariate specific and indicate departures for the expected value of a covariate x_k at failure time t_i . Violations of the proportional hazards assumption can be detected by assessing whether the Schoenfeld residuals for a given covariate are correlated with time. Any significant correlation indicates that a covariate’s effect changes over time and therefore violates the proportional hazards assumption.

It is also possible to assess departures from the proportional hazards assumption graphically by plotting the Schoenfeld residuals against time. Any visual pattern in the residuals with respect to time indicates a violation of the proportional hazards assumption. These techniques can be implemented with semiparametric cure models by using the modified Schoenfeld residuals developed for use with cure models by Peng and Taylor (2017) and Wileyto et al. (2013). Covariates that do not meet this assumption may be dealt with in the usual ways, i.e., by stratifying on the offending covariate or by incorporating interactions with the log of time (Peng, 2003).

The PHCM has been extended to accommodate a number of other issues that are common with survival data. Stratification can be used to allow the conditional baseline hazard to vary by groups of observations (Peng, 2003). Non-linear effects may also be incorporated using the univariate transformations of the covariates (see Therneau and Grambsch, 2000b). Variants of the semiparametric proportional hazards mixture model have been developed that can accommodate interval-censored data (Liu and Shen, 2009; Hu and Xiang, 2013; Lam, Wong, and Zhou, 2013), frailty terms (Price and Manatunga, 2001; Peng and Zhang, 2008a; Peng and Zhang, 2008b), and covariate and time-dependent censoring (Lu and Ying, 2004; Othus, Li, and Tiwari, 2009).

2.4 Software

At present, several options exist for estimating the PHCM in R, including the **intercure**, **mixcure**, **rcure**, and **smcure** packages (Brettas, 2016; Cai et al., 2012; Han, Zhang, and Shao, 2017; Peng and Taylor, 2017). Although each of these packages has their advantages, none of the them are able to fit models on data with time-varying covariates. This is an important limitation, as it limits researchers to analyzing purely cross-sectional datasets.

To fill this gap, I developed the **tv cure** R package. The package allows for estimating the PHCM using time-varying covariates using the standard syntax of the **survival** package (Therneau and Grambsch, 2000a). Estimation is performed using the expectation maximization algorithm described in Appendix A.1. The package currently supports the use of logit or probit models for the cure equation. Because the results of the cure equation are prone to experience quasi-complete separation, I also include the functionality to allow the generalized linear model to be estimated using biased-reduced generalized linear models (such as Firth's (1993) bias-reduced logistic regression).using the **brglm** package (Kosmidis, 2020). Standard errors for the coefficients are estimated using a nonparametric bootstrap with stratified random sampling. The package supports the use of parallel processing through the **foreach**, **snow**, and **doSNOW** (**microsoftcorporation2019**; Microsoft and Weston, 2020; Tierney et al., 2018) packages.

In addition, the **tvcore** package includes several functions designed to facilitate the interpretation and presentation of results. The package includes a prediction function for computing and plotting various quantities of interest for different covariate profiles, including the probability of failure, probability of cure, conditional survivor function, conditional baseline survivor function, and the marginal survivor population. At present, these functions are not capable of estimating confidence intervals for these quantities. However, the functionality to do so using simulation-based methods, as employed by Beger et al. (2017), is currently in development. In addition, a function designed to help produce publication ready tables in conjunction with the **xtable** package is included (Dahl et al., 2019). These functions were used to create all results tables and plots presented in the replication analysis.

2.5 Applying Cure Models to International Relations

The problem of cured observations is common in international relations. Many of the phenomena studied only affect a portion of the observations studied over the long term, especially when dealing with rare events such as war onset, assassinations, and coups. While the use of survival analysis can and should be used to model these phenomena when appropriate, using these models without regard for their underlying assumptions undercuts the validity of the results obtained.

Some analysts attempt to address this by removing subjects from the sample using heuristic shortcuts. The most common example of this in international relations is the use of politically relevant dyads, which are typically defined as pairs of states that are contiguous or contain at least one major power. Scholars often assume that phenomena of interest, such as military disputes, are highly unlikely to occur within non-relevant dyads and use this as a justification to remove those dyads from the sample. Unfortunately, the use of politically relevant dyads as a criteria does not cleanly divide dyads that are susceptible from those that are not. As Lemke and Reed (2001) point out, many non-relevant dyads experience militarized disputes, while many politically relevant dyads are extremely unlikely to go to war.

For this reason, it is better to model the probability that states are susceptible to war and use this model to correct the estimates of the hazard equation accordingly. Xiang (2010) shows that this approach

is superior in the context of binary response models. He demonstrates that the results of an analysis of all dyads using a split population probit model outperforms the use of a simple probit using politically relevant dyads when analyzing militarized interstate dispute onset.

The other major advantage of cure models is their ability to help test theoretical arguments that distinguish between factors that affect susceptibility and event onset. The underlying data-generating process assumed by cure models maps well onto certain theoretical constructs that are frequently used in international relations. For example, theories that distinguish between the underlying and proximate causes of a phenomena reflect the logic underlying cure models. For example, Vasquez (2009) distinguishes between the underlying and proximate causes of war, and Belkin and Schofer (2003) theorize about the structural and proximate causes of coups (see also Beger et al., 2017). These theories typically assume that some subjects are structurally predisposed to experiencing an event, but do not actually experience the event until a proximate cause triggers the event. According to this logic, both underlying and proximate causes must be present in order for the event to occur. Cure models capture the data-generating process underlying the logic of structural and proximate causes well. Whereas structural causes influence whether an individual is at risk or cured, proximate causes affect the timing and onset of an event.

Others have argued that forecasting models of intrastate conflict need to incorporate both structural and proximate causes. This is due to the fact that causes of instability (e.g., mass protests) may have little chance of precipitating civil conflict in states that are structurally unlikely to experience conflict but may have a very large effect in those that are predisposed. However, existing attempts to forecast conflict events using a combination of structural and proximate causes have still been found wanting (Tikuisis, Carment, and Samy, 2013). The use of cure models to account for structural causes of instability using the cure equation and the proximate causes of instability using the hazard equation may represent one avenue by which scholars could attempt to improve the forecasting of such events.

Cure models may also be useful in testing theories that distinguish between short-term and long-term effects. One example involves factors that decrease the probability of civil war recurrence (see e.g., Braithwaite and Sudduth, 2016; Collier, Hoeffler, and Soderbom, 2008; Hartzell and Hoddie, 2003; Joshi

and Mason, 2011; Loyle and Appel, 2017; Mason et al., 2011; Mason and Greig, 2017; Quinn, Mason, and Gurses, 2007; Rustad and Binningsbø, 2012; Walter, 2004; Walter, 2015). Although roughly 30-50 percent of countries experience renewed fighting after civil conflicts (depending on the data used), most do not. Theoretically, while some variables may influence the probability of permanent peace within a country, others may have temporary effects and simply extend the time until a war recurs. In most cases, however, what is really of interest is whether a variable affects the probability that a conflict occurs altogether. Standard survival models merely demonstrate that a variable influences the time until war recurs, which may be due to either a short or long-term effect. By contrast, cure models can be used to assess whether a variable influences the time until war recurrence as well as whether a country is “cured” of war.

2.6 Replication Analysis of Civil Conflict Recurrence

To illustrate how standard Cox models and the PHCM can produce different results, I replicate an analysis of the duration of peace after civil conflict by Loyle and Appel (2017). As discussed above, civil conflict recurrence is a prime example of a phenomenon which not all subjects will experience. Loyle and Appel (2017) examine the recurrence of civil conflict during the period 1950-2006 using data from the UCDP/PRIO Armed Conflict Dataset (Gleditsch et al., 2002) and the UCDP Conflict Termination Dataset (Kreutz, 2010). The conflict termination data divides the internal conflicts contained in the UCDP data into episodes based on the extent of the fighting in each year. Episodes of civil conflict must entail fighting between a country’s government and at least one armed opposition group and must produce at least 25 battle-related deaths a year. Conflict episodes begin in the year that fatalities first exceed 25 deaths and end when fatalities drop below this threshold.

The dependent variable is the time between the end of a conflict episode and the beginning of a new episode of the same conflict. The unit-of-analysis is the post-conflict-episode country-year (since countries may be involved in multiple civil conflicts at one time, there may be multiple observations for each country-year). Subjects enter the dataset following the end of a civil conflict episode and remain until

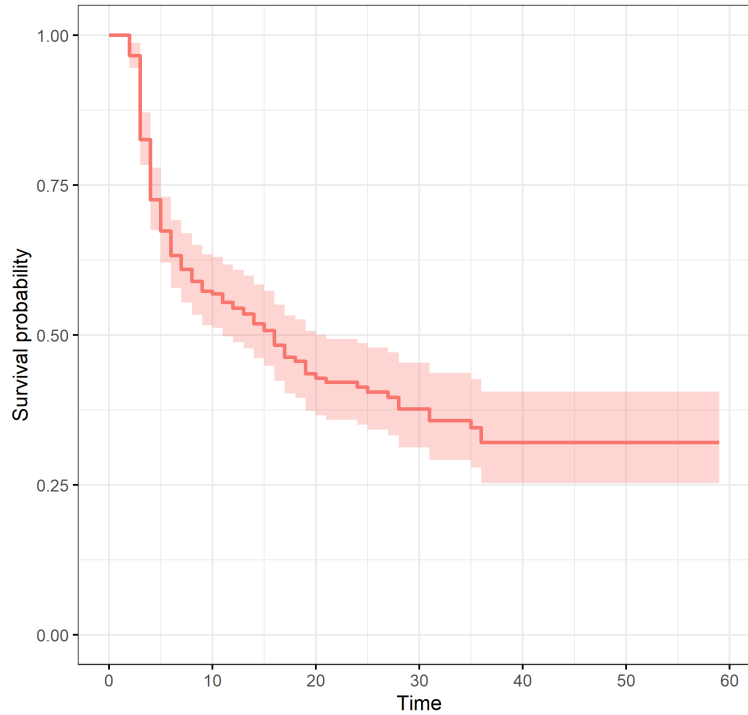


Figure 2.1: Kaplan-Meier Survival Estimate for Civil Conflict Recurrence

the same conflict produces another episode of fighting (i.e., failure) or are right-censored in 2006. The dataset used contains data on a total of 297 conflict episodes, of which 154 recur.

Figure 2.1 plots the nonparametric Kaplan-Meier survival estimate for time to conflict recurrence. After 57 years, an estimated 30 percent of cases have yet to produce another conflict, indicating that many cases are unlikely to experience conflict recurrence. This provides additional evidence that the use of a cure model is necessary to accurately model the data-generating process.

2.6.1 Independent Variables

The theoretical question Loyle and Appel (2017) address concerns the effect of post-conflict justice (PCJ) processes on conflict recurrence. They argue that PCJs reduce the probability of conflict recurrence by either decreasing the motivations for rebellion or decreasing the opportunity to successfully rebel. Motivation-focused PCJ processes include measures such as reparations, amnesty, truth and reconciliation

commissions, and judicial proceedings that include government agents accused of wrongdoing. These measures are intended to address the grievances that former rebels or other individuals may harbor against the government due to its conduct during the conflict. By contrast, opportunity-reducing mechanisms are aimed primarily at deterring potential rebels from taking up arms or otherwise undercutting the ability of these groups to effectively fight against the government. These include processes such as trials directed solely at the opposition, exiling members of the opposition group, and purging politicians or other members of society that threaten the incumbent government.

Using data from the Postconflict Justice dataset (Binningsbø et al., 2012), Loyle and Appel (2017) construct indices for the number of motivation and opportunity-reducing PCJ processes implemented in the five years following each conflict episode. The motivation index includes one point each if the government implements reparation programs designed to address losses caused by the conflict, amnesty provisions, and trials that include government agents. The opportunity index includes one point each for the use of exiles, purges, or trials directed solely at the opposition. Loyle and Appel (2017) find that motivation-decreasing processes reduce the probability of conflict recurrence while opportunity-decreasing processes do not. I examine the robustness of these findings by estimating a Cox and cure model and comparing the results. Before doing so, it is necessary to specify which variables will be included in each equation of the cure model.

Based on Loyle and Appel's findings, a potential follow-up question may be whether these policies have short or long-term effects. For example, it is possible that motivation-decreasing PCJ processes may temporarily resolve grievances of individuals against the government due to its actions during the conflict without resolving the underlying issue that motivated the conflict. In this scenario, these policies would be associated with an increased duration of peace but would not reduce the probability of eventual conflict recurrence. In order to address this question, I include the motivation and opportunity PCJ indices to both equations. This allows me to examine whether these policies had short or long-term effects on the probability of conflict recurrence.

Loyle and Appel (2017) also include a litany of control variables related to the recurrence of civil conflict. The control variables are assigned to each equation as follows: First, like PCJ processes, the implementation of power-sharing provisions and peacekeeping operations are policy interventions that may have short or long-term effects. As such, I include them in both equations. Power-sharing is measured using a dummy variable for whether a power-sharing agreement was implemented in the post-conflict state (Hartzell and Hoddie, 2003; Harbom, Hogbladh, and Wallensteen, 2006; Loyle and Appel, 2017; Mattes and Savun, 2010). Peacekeeping is measured using a dummy variable for whether peacekeepers were present in the country in a given year.

I include two additional variables in the hazard equation that speak to changing conditions that may create windows of volatility during which civil conflict may be especially likely. First, Loyle and Appel (2017) use the number of military personnel controlled by the government to control for variations in rebels' opportunity to challenge the government over time. Data comes from the Correlates of War National Military Capabilities dataset (Singer, Bremer, and Stuckey, 1972; Singer, 1987). To control for grievances related to changing economic conditions, Loyle and Appel (2017) include a measure for growth in real gross domestic product (GDP) per capita using data from Gleditsch (2002).

I include the following three variables in the cure equation to account for fixed, structural characteristics that create the potential for grievances to intensify or reemerge in the post-conflict environment: the log of GDP per capita (Gleditsch, 2002), whether the conflict occurred over an ethnic issue (Gleditsch et al., 2002), and whether a country is a democracy. Democracy is measured using a dummy variable for whether a post-conflict state has a Polity score above five (Marshall and Jaggers, 2002; Marshall and Jaggers, 2013).

I include five variables related to the previous conflict and its termination in the cure equation: the log of conflict duration (Binningsbø et al., 2012), the log of battle deaths per capita, a dummy variable for whether the conflict ended in victory (Kreutz, 2010), the number of rebel groups active during the conflict Gleditsch et al. (2002), and whether the conflict ended in a peace agreement (Kreutz, 2010). The first three variables speak to the information that a conflict conveyed about the strength of the government

and the relative capabilities of the disputants. These qualities are fixed with respect to time and act as a constraint on the opportunity for rebel groups to effectively challenge the government. The number of rebel groups speaks to the number of organized groups that could potentially attempt to challenge the status quo in the post-conflict environment. Conflicts that ended with a peace agreement are more likely to last by resolving disputed issues and/or creating mechanisms that prevent the combatants from engaging in renewed fighting. Finally, the Cold War dummy variable is related to the opportunity for rebels to successfully wage military campaigns in the post-Cold War environment.

2.6.2 Substantive Results

Table 2.1 presents the results of the analysis. Model 1 is a standard Cox proportional hazard model. Model 2 is a PHCM including select variables in each equation based on the logic outlined above. For the sake of comparison, Model 3 includes all variables in both equations. The Akaike Information Criteria (AIC) for Model 2 is 2,977, while it is 2,959 for Model 3. The fact that the AIC is lower for Model 3 indicates that the fully specified model performs better than Model 2 in this case. I conducted proportional hazards tests for each model and found that none of the models required proportional hazards corrections.

The entries in Table 2.1 for Model 1 are hazard coefficients. Positive values indicate that a variable is positively associated with an increased hazard rate, and therefore, decreased survival times. The results for Models 2 and 3 are split across two columns. The first column contains the logistic regression coefficients for the model of whether a country is susceptible to civil war recurrence. Positive coefficients indicate that a variable is positively correlated with a higher likelihood of repeat civil war. The second column of results for Models 2 and 3 contains the hazard coefficients that describe the effect of variables on the survival times of susceptible subjects. The interpretation of these coefficients is the same as in Model 1: positive coefficients indicate that a variable is associated with an increased hazard rate.

I begin by discussing the results with respect to Loyle and Appel's (2017) primary independent variables. In a general sense, the results of all three models indicate that motivation-decreasing PCJ processes increase the duration of post-conflict peace while opposition-decreasing PCJ processes do not. The esti-

Table 2.1: Models of Civil Conflict Recurrence

	Model 1	Model 2		Model 3	
	Hazard Coef.	Logit Coef.	Hazard Coef.	Logit Coef.	Hazard Coef.
Motivation Post-conflict Justice	-0.586* (0.2)	-0.309 (0.22)	-0.163* (0.044)	-0.371 (0.216)	-0.168* (0.055)
Opportunity Post-conflict Justice	-0.098 (0.173)	-0.139 (0.173)	0.02 (0.05)	-0.137 (0.18)	0.007 (0.063)
Power Sharing	-0.605 (0.45)	-0.426 (0.502)	-0.107 (0.879)	-0.451 (0.451)	-0.058 (0.113)
Peacekeeping	-0.94* (0.39)	-0.806 (0.47)	-0.129 (0.076)	-0.888* (0.433)	-0.083 (0.087)
Military Personnel	-0.366* (0.125)		-0.139* (0.041)	-0.425* (0.104)	-0.012 (0.042)
GDP Growth	-1.222 (0.93)		-0.47 (0.442)	-0.577 (0.92)	-0.217 (0.309)
ln GDP per Capita	-0.141 (0.102)	-0.286* (0.102)		-0.211* (0.103)	0.06 (0.04)
Ethnic War	0.093 (0.198)	0.033 (0.221)		0.063 (0.21)	0.031 (0.061)
Democracy	-0.157 (0.234)	-0.07 (0.231)		-0.078 (0.227)	0.02 (0.078)
Conflict Duration	-0.064 (0.117)	-0.179 (0.121)		-0.032 (0.126)	-0.054 (0.035)
Battle Deaths per Capita	-0.039 (0.048)	-0.007 (0.043)		-0.07 (0.046)	0.02 (0.015)
Victory	-0.826* (0.233)	-0.773* (0.189)		-0.949* (0.211)	-0.008 (0.102)
Peace Agreement	0.336 (0.348)	0.293 (0.414)		0.181 (0.418)	0.007 (0.084)
Number of Rebel Groups	0.968* (0.118)	1.051* (0.121)		1.036* (0.118)	0.066* (0.032)
Post-Cold War	0.502* (0.194)	0.506* (0.187)		0.377* (0.18)	-0.012 (0.095)
Intercept		1.771 (2.216)		1.982 (2.248)	
Number of Observations	3773		3773		3773
Number of Failures	154		154		154

Note: Standard errors in parentheses. Standard errors for Model 2 were estimated using 500 bootstrap replications. * $p < 0.05$.

mated coefficients for motivation-decreasing processes produced by both models are negative and significant, indicating that those processes are associated with a decreased hazard rate. However, the fact that motivation-decreasing processes do not have a significant effect on the probability that a subject is cured or not provides additional detail about the nature of the effects of motivation-decreasing processes. Specifically, these processes appears to forestall the recurrence of civil conflict but do not prevent its eventual recurrence.

In addition, the size of the substantive effects differs between the three models. The substantive effect of Model 1 is much larger than that indicated by Models 2 and 3. Figure 2.2 plots the population survival curves for both Model 1 and 2 when zero motivation-decreasing PCJ processes are implemented in a post-conflict country versus when three processes are implemented (all other variables are held constant at their medians). Comparing these two graphs demonstrates that the relative effect of motivation-decreasing PCJ processes is much larger for Model 1 than for Model 2. For Model 1, the maximum expected difference in the probability of survival for countries that implement zero and three PCJ processes is roughly 0.45. By contrast, the maximum difference between the two curves produced by Model 2 is roughly 2 percent. Moreover, the survival curves in Model 2 converge after 36 years, indicating that there is no longer any difference in the probability between the two subjects. This is to be expected, since only cured subjects remain in the sample at this point. The results of Model 1 thus imply that the substantive effect of PCJ processes are much larger than the results of Model 2 do. When evaluating the differences between the two models above, it is natural to ask which model to believe. Since there are strong theoretical reasons to believe that the cure model describes the data-generating process for civil-conflict much better than the Cox model, we should expect, a priori, that the cure model's results are more reliable.

The results with respect to military personnel display the same pattern as motivation-decreasing PCJ processes when comparing Models 1 and 2. Both coefficient estimates are negative and significant, indicating that subjects are more likely to experience conflict recurrence when their military personnel is low. To compare the results, Figure 2.3 plots the survival curves for each model at the fifth and ninety-fifth percentiles for military personnel. As before, the survival function produced by the Cox model implies

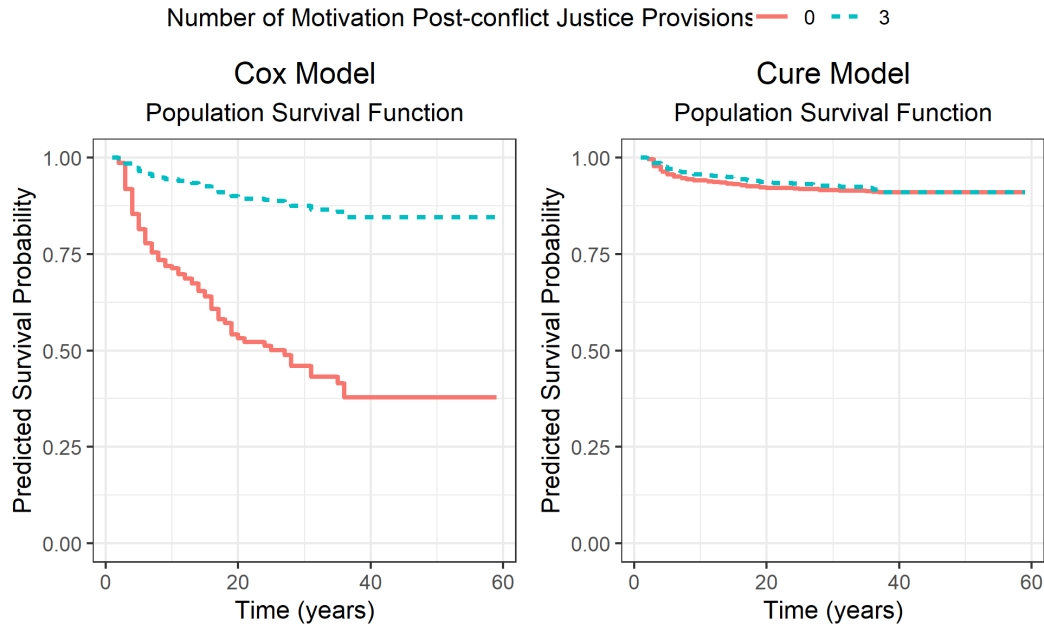


Figure 2.2: Effects of Motivation-Decreasing Post-Conflict Justice Processes on Conflict Recurrence

that military personnel has a large substantive effect: after 36 years, an average subject with a low number of military personnel is nearly 42 percentage points more likely to experience another conflict than one with a large number of personnel. However, the right-hand survival plot shows that the effect is predicted to be much smaller when a cure model is used. An average case with a low number of military personnel is never more than 2 percentage points more likely to experience conflict onset than one that does not.

Notably, Model 3 implies a slightly different result. Instead of being significant in the hazard equation, military personnel are significant in the cure equation. This indicates that states with lower military personnel are more likely to be susceptible to repeat civil war, but susceptible states will not experience civil war more quickly if they have less military personnel. The predicted probability when military personnel is at the fifth percentile is 0.03, while it is 0.01 for subjects with military personnel

Another result worth highlighting is that peacekeeping is significant in Model 1 but is not significant in either equation for Model 2. The estimated coefficient of -0.94 indicates that peacekeepers are associated with a decrease in the hazard rate of conflict recurrence and an increase in the probability of survival. To

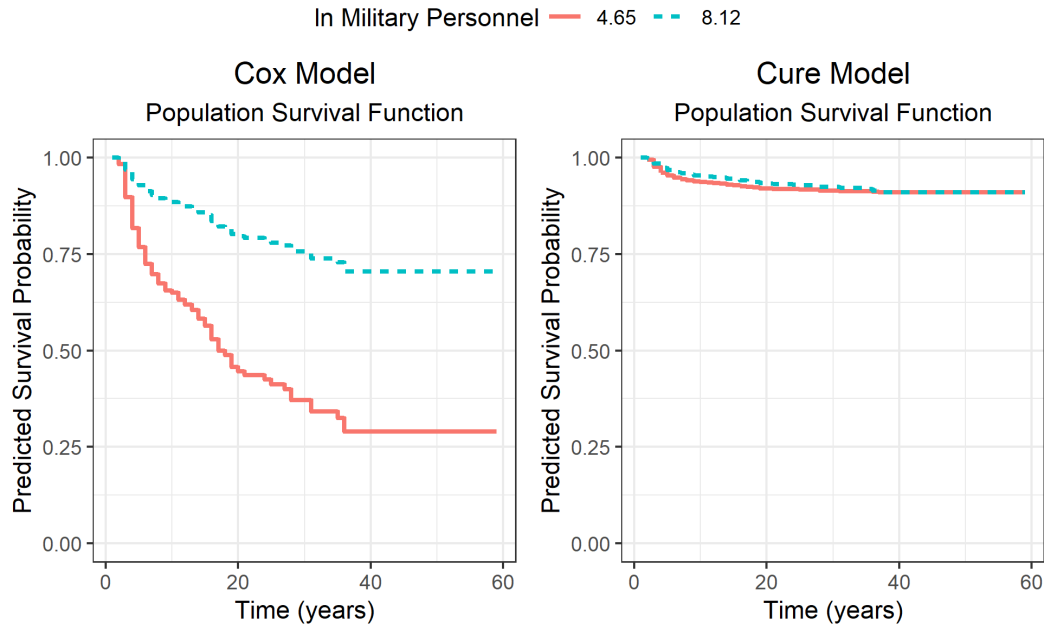


Figure 2.3: Effects of Military Personnel on Timing of Conflict Recurrence

assess the substantive effect of peacekeeping produced by Model 1, Figure 2.4 plots the survival probability when peacekeepers are present and when they are not. The effect-size is fairly large: After 36 years, the predicted survival probability when peacekeepers are present is 0.68, while it is only 0.38 when they are not. Each model thus implies very different results. Whereas Model 1 indicates that there is a large substantive effect, Model 2 indicates that we cannot be confident that peacekeepers have any effect, either on the short-term probability of survival or the overall probability that a case is susceptible to begin with.

Another difference can be seen between the results with respect to the variable for GDP per capita. Although the estimated coefficient for Model 1 is insignificant, the coefficient in the cure equation of Model 2 is negative and significant. This indicates that countries with high income levels are less likely to be at risk for additional conflicts. To assess the substantive effects, Figure 2.5 plots the predicted probability that a country is susceptible to another conflict across the range from the fifth to the ninety-fifth percentile of GDP. At the lowest value (roughly 537,000,000 dollars), the probability that a state is susceptible to additional conflicts is 0.13, while at the highest value (10,800,000,000 dollars), the probability of additional

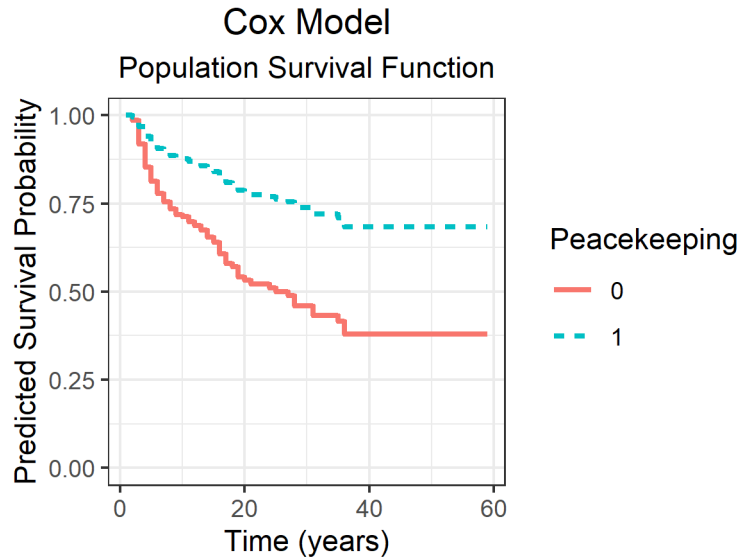


Figure 2.4: Effect of Peacekeeping on Timing of Conflict Recurrence

conflicts is 0.06. There is thus a seven percentage point difference in the likelihood that an average country is susceptible to conflict in a given year between high-wealth (relative to the sample) and low-wealth countries. The fact that higher-income countries are less likely to experience civil war is a robust findings in the literature (Dixon, 2009). As such, the fact that GDP behaves as expected in Model 2 but not in Model 1 lends additional face validity to the notion that results of Model 2 should be preferred.

The remaining variables that are significant in Model 1 (victory, the number of rebel groups, and post-Cold War) are also significant in the cure equation of Model 2. The estimated coefficients for victory are negative and significant in both models. The substantive interpretation of each variable differs, however. The negative coefficient of -0.83 in Model 1 indicates that victory is associated with a lower hazard rate, while the negative coefficient of -0.77 indicates a lower probability of failure for Models 1 and 2, respectively. When a variable is significant in the Cox model and the cure equation of the PHCM, it is difficult to directly compare the relative size of the substantive effects across the two models. The coefficients of the two models are interpreted differently, as are survival probabilities and the probability of being cured. It may thus only be possible to make very general statements about effect size if, for example, the difference

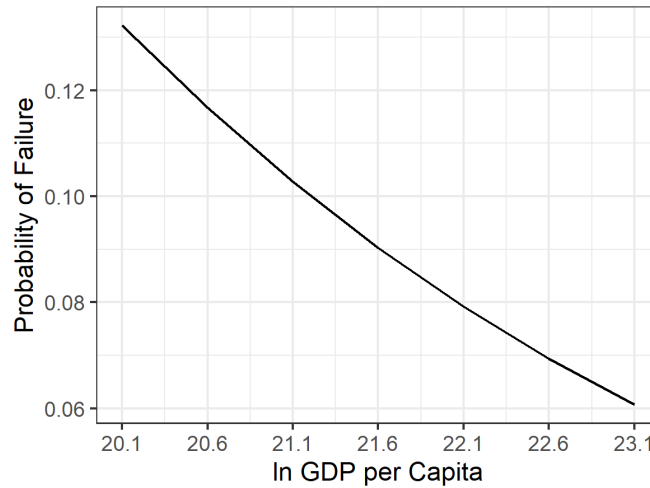


Figure 2.5: Effect of GDP per Capita on Susceptibility to Conflict Recurrence

in survival probability is very large, while the difference in the predicted probability of the logit model is very small.

To assess the substantive effects of Model 1 individually, Figure 2.6 plots the survival curve for cases which end in victory and those that do not. The difference between the predicted survival probability is relatively large. After 36 years, the probability of survival is roughly 0.38 when a conflict had a clear victor, but only about 0.24 for cases that did not. For Model 2, the predicted probability of failure is 0.09 for cases where a conflict ended in victory but a 0.18 probability of failure when it did not. Thus, the results of Model 2 indicate that there is a 0.09 change in the probability that a post-conflict country is susceptible to a civil war in a given year.

For rebel groups, the coefficient estimate has a significant value of 0.97, indicating that more rebel groups increase the probability of future rebellions. Figure 2.7 plots the survival curves for conflicts that involved one-four rebel groups. The difference in the probability of survival between conflicts involving one rebel group (the median) and two rebel groups is 30 percent after 30 years. Further, a conflict that involves four rebel groups only has a two percent chance of surviving five years, and conflicts involving three or four rebel groups are virtually guaranteed to eventually fail. The results of Model 2 also show

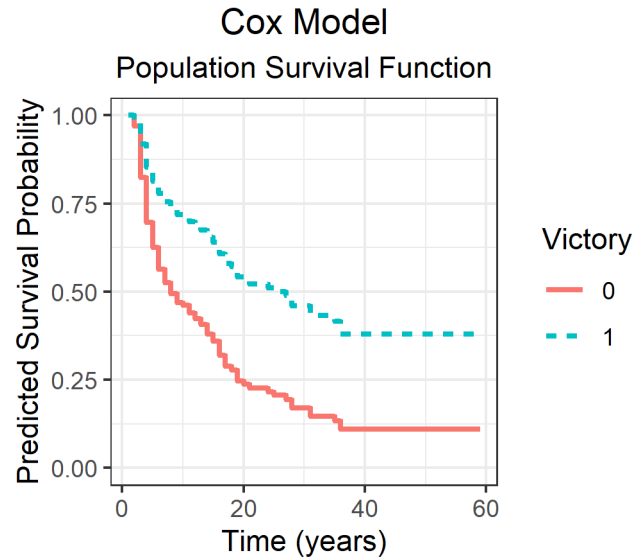


Figure 2.6: Effect of Victory on Timing of Conflict Recurrence

that rebel groups have a large effect. The predicted probability of failing in a given year when there is one rebel group is 0.09, while the predicted probability of failing when there are two is 0.21, a difference of 12 percentage points. When there are 4 rebel groups, the predicted probability of failing in a given year is 0.70.

The variable for post-Cold War is also positive and significant for both models. The positive hazard coefficient of 0.50 produced by Model 1 indicates that conflicts fail more quickly after the Cold War, while the positive coefficient of 0.51 for Model 2 indicates that conflicts that occur after the Cold War are more susceptible to failure. Figure 2.8 plots the predicted survival curves for conflicts that occur during and after the Cold War. The results show that the timing of conflict has a modest effect on the probability of survival. After 36 years, the survival probability for a conflict during the Cold War 0.56, while the survival probability for conflicts afterwards is 0.38. For Model 2, the probability of failure prior to the Cold War is 0.06, while the probability of failure is 0.09. The effect size produced by Model 2 thus appears to be much smaller.

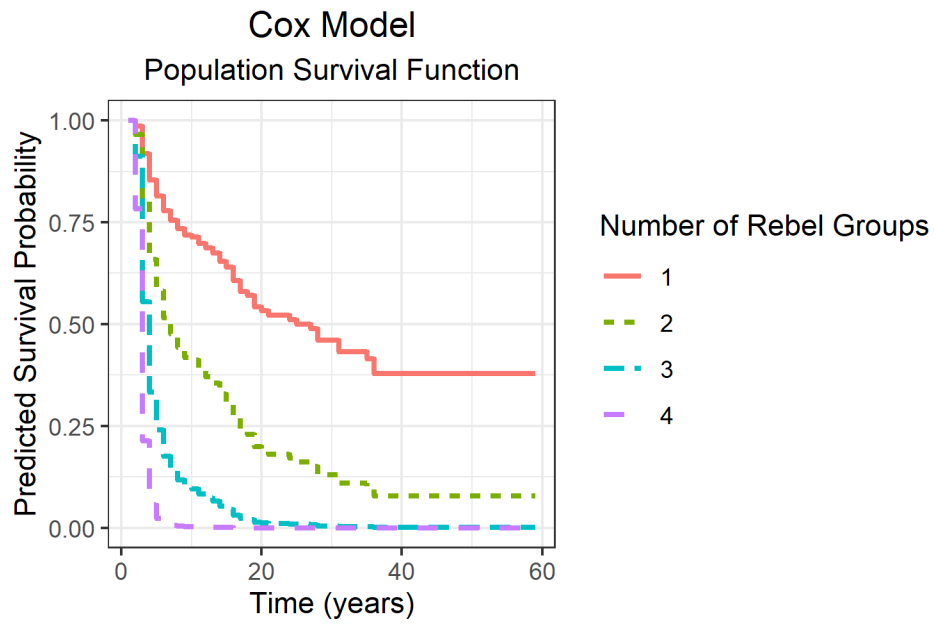


Figure 2.7: Effect of Number of Rebel Groups on Timing of Conflict Recurrence

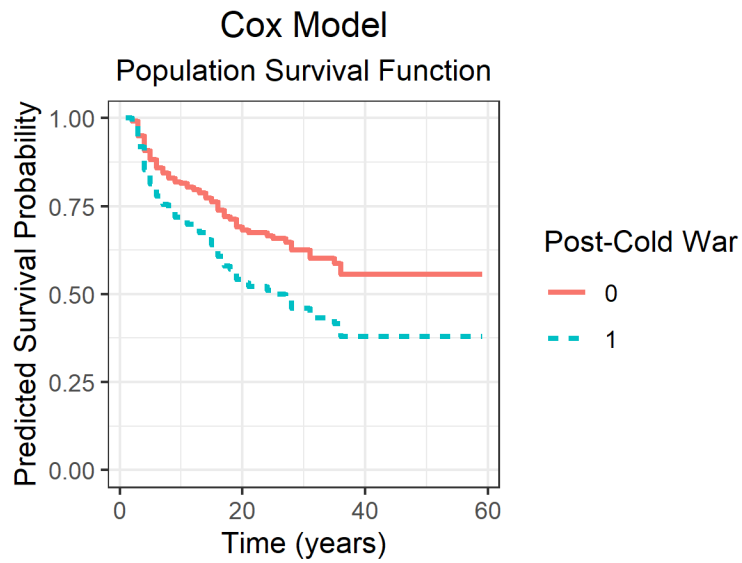


Figure 2.8: Effect of Post-Cold War on Timing of Conflict Recurrence

2.7 Conclusion

In this chapter I introduced readers to the proportional hazards cure model. Unlike traditional survival models, cure models account for the fact that some subjects may not experience an event. In doing so, they correct for the potential bias and inconsistency that can result from ignoring this assumption. Given that many international phenomena are only likely to occur between a small subset of countries, I argue that scholars of international relations would be well-served by using these models more frequently. In addition, this article introduces the **tv cure R package** I developed to fit the PHCM in R. This new package expands analysts' ability to apply the PHCM by allowing them to incorporate data that includes time-varying covariates.

I demonstrate the utility of the PHCM by replicating an analysis of civil conflict recurrence by Loyle and Appel (2017). The results of this analysis illustrate three generalizable differences that can result when using a Cox model when a cure model is more appropriate. First, the use of a standard Cox model can lead analysts to conclude that a variable has a significant effect when there is not (i.e., Type I errors). The results above provide an example of this in the form of the peacekeeping variable. Whereas the Cox model would lead analysts to conclude that there is a significant effect, the cure model does not support this finding.

Second, using a standard Cox model can lead analysts to conclude that a variable has an insignificant effect when the cure model would find that there is a significant effect (i.e., Type II errors). This is illustrated by the results each model produced with respect to the GDP per capita variable above. Whereas the Cox model did not detect a significant effect for this variable, the cure model found that it did have a significant effect on the probability of being susceptible or cured. This provides an even stronger substantive result than simply finding a significant effect in the cure model by implying that average income makes countries less likely to experience civil conflict altogether rather than simply delaying it. Third, even when both models produce significant results, the substantive effects produced by both models may differ substantially. This is exemplified by the results with respect to motivation-decreasing PCJ processes

and military personnel. In both cases, the Cox model predicted much greater observable differences than the cure model did.

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APPENDIX A

SUPPLEMENTARY MATERIALS FOR

CHAPTER 2

A.1 Estimating Semiparametric Cure Models using Expectation Maximization

Since the distribution of S_u is left unspecified, maximizing the full likelihood of the semiparametric cure model using standard Newton-Raphson type algorithms is not possible. Standard Cox proportional hazards models make use of the partial likelihood method to eliminate S_u from the likelihood. However, due to the more complex form of the semiparametric PH cure model, this is not possible.

As such, semiparametric cure models require an estimation technique that can maximize the full-likelihood and provide estimates of S_u . Peng and Dear (2000) and Sy and Taylor (2000) suggest using an expectation-maximization (EM) algorithm.¹ The complete data log-likelihood is given by

$$\mathcal{L}_C(\beta, \gamma, H_0) = \prod_{i=1}^n p_i^{y_i} (1 - p_i)^{1-y_i} \prod_{i=1}^n \{h_u(t_i)^{\delta_i y_i} S_u(t_i)^{y_i}\}, \quad (\text{A.1})$$

¹Alternatively, the PHMC can be estimated using Markov Chain Monte Carlo simulations (Kuk and Chen, 1992), multiple imputation (Lam, Fong, and Tang, 2005), and Bayesian techniques.

where the first product term contains the parameters related to the incidence component of the model and the second contains the parameters related to the latency component. The log of Equation A.1 can be expressed as the sum of two likelihood functions:

$$\mathcal{L}_1(\beta) = \sum_{i=1}^n \left\{ y_i \log(p_i) + (1 - y_i) \log[1 - p_i] \right\}, \quad (\text{A.2})$$

$$\mathcal{L}_2(\gamma, S_u) = \sum_{i=1}^n \left\{ y_i \delta_i \log h_u(t_i) + y_i \log[S_u(t_i)] \right\}. \quad (\text{A.3})$$

The E-Step of the EM algorithm takes the conditional expectation of the complete log-likelihood function with respect to the unobserved y_i values for the given current estimates of β , γ , and S_{u0} .² Although y_i is not observed, the expectation of y_i conditional on the observed data and current parameter estimates is sufficient to conduct this step since Equations A.2 and A.3 are linear functions of y_i . Let $w_i^{\{m\}}$ denote the conditional expectation of y_i given the current parameter estimates $\Theta^{\{m\}} = (\beta^{\{m\}})$, given by

$$w_i^{\{m\}} = E(y_i | \delta_i, t_i, \mathbf{x}_i, \mathbf{z}_i) = \delta_i + (1 - \delta_i) \left(\frac{p_i S_{u0}(t_i)}{1 - p_i + p_i S_{u0}(t_i)} \right). \quad (\text{A.4})$$

For uncensored individuals ($\delta_i = 1$), the value of y_i is known and $w_i^{\{m\}}$ reduces to 1. For censored individuals, $w_i^{\{m\}}$ reduces to the probability of the i^{th} censored individual being uncured. Put otherwise, for censored individuals, $w_i^{\{m\}}$ “represents a fractional allocation to the susceptible group,” (Sy and Taylor, 2000, p. 229). Substituting $w_i^{\{m\}}$ for y_i in Equation A.2 and Equation A.3 produces

$$\mathcal{L}_1(\beta) = \sum_{i=1}^n \left\{ w_i^{\{m\}} \log(p_i) + (1 - w_i^{\{m\}}) \log[1 - p_i] \right\}, \quad (\text{A.5})$$

$$\mathcal{L}_2(\gamma, S_u) = \sum_{i=1}^n \left\{ w_i^{\{m\}} \delta_i \log h_u(t_i) + w_i^{\{m\}} \log[S_u(t_i)] \right\}. \quad (\text{A.6})$$

The M-step involves maximizing the log-likelihood functions of Equation A.5 and Equation A.6 with respect to the unknown parameters β , γ , and S_{u0} using the current values of $w_i^{\{m\}}$. Since Equation

²Initial estimates for w_i are derived by setting all censored cases to 0 and all uncensored cases to 1.

A.5 does not depend on the value of β or S_{u0} , estimates of γ can be obtained by maximizing Equation A.5 using standard binomial regression routines. Similarly, since Equation A.6 does not depend on the value of γ , estimates of β and S_{u0} can be obtained by maximizing Equation A.6 using standard Cox PH routines (Peng, 2003; Cai et al., 2012), where S_{u0} is estimated using Breslow (1972)'s version of the Cox PH model, given by

$$\hat{S}_0(t|Y = 1) = \exp \left(- \sum_{j:t_{(j)} \leq t} \frac{d_{t_{(j)}}}{\sum_{i \in R(t_{(j)})} w_i^{\{m\}} \exp(\mathbf{x}_i' \hat{\beta})} \right), \quad (\text{A.7})$$

where $d_{t_{(j)}}$ is the number of events at time $t_{(j)}$ and $R(t_{(j)})$ is the set of observations that are at risk of failure at $t_{(j)}$.³

Once estimates of β have been obtained, estimates of the conditional baseline survival function, S_{u0} , are obtained using profile likelihood methods. This typically involves using a modification of Breslow (1972)'s likelihood for the Cox PH model.⁴ Once the estimates of β , γ , and S_{u0} are obtained, the E-step is repeated using the newly obtained estimates to re-estimate the value of $w_i^{\{m\}}$. Estimation proceeds by iterating between the E and M steps until the values of the parameters converge. Fang, Li, and Sun (2005) demonstrate that the maximum likelihood estimates of S_u are consistent and asymptotically normally distributed.

The estimator defined in Equation A.7 may not approach zero for t greater than the maximum observed failure time, $t_{(k)}$. Taylor (1995) characterizes this as an identifiability problem in which the tail of the S_u distribution is difficult to estimate. Taylor (1995) suggested imposing the constraint that $S_{u0} = 0$ for $t > t_{(k)}$. This constraint is achieved by setting $w_i^{\{m\}} = 0$ for observations where $t > t_{(k)}$ in the E-step. This effectively eliminates the identifiability problem and leads to more stable parameter estimates and faster convergence (Taylor, 1995). In addition, this constraint may lead to less biased estimates of β and γ in the presence of high levels of censoring. Substantively, this constraint implies that most of the subjects

³This constitutes using a modified version of the Nelson-Aalen estimator to estimate the baseline cumulative hazard and then estimating the survivor function using $S_{u0} = \exp -H_0(t|\hat{Y} = 1)$.

⁴Sy and Taylor (2000) demonstrate that S_u can also be estimated using the nonparametric full likelihood method of Kalbfleisch and Prentice (1980).

left at the end of the observation period are members of the cured group and are unlikely to fail in the future. Put otherwise, the use of the semiparametric PH mixture cure model may not be appropriate when the follow-up period of a study is not long enough for most of the susceptible individuals to have already failed.

Because cure models tend to be demanding on the data, they are prone to issues of non-convergence or unstable coefficient estimates in small samples or in samples in which there are very few failures or very few cured observations (although the constraint on the survivor function discussed above improves the performance of the cure model in this regard (Taylor, 1995; Sy and Taylor, 2000)). In addition, cure models may be prone to complete or quasicomplete separation (i.e. infinite coefficient and variance estimates) in either the incidence or latency components. This is particularly likely in small samples and when there are very few failures or very few cured subjects (Sy and Taylor, 2000). Although this is difficult to deal with without omitting covariates from the model, the use of Bayesian priors to ameliorate these issues is an area of active research (Han, Zhang, and Shao, 2017).

Since estimates of the variance of the estimated parameters are not directly available from the EM algorithm, alternate methods of estimating the standard errors are necessary for hypothesis testing.⁵ As such the standard errors of the coefficients are typically estimated using a nonparametric bootstrap with replacement to (e.g. Peng, 2003; Cai et al., 2012).

⁵Although scholars have derived analytical approximations of the standard errors, including Peng and Dear (2000), Sy and Taylor (2000), Fang, Li, and Sun (2005), and Xu, Baines, and Wang (2014), these estimates tend to be unstable and are difficult to estimate when more than a few covariates are included. In addition, these formulas cannot be easily be adapted to accommodate variations or extensions of the semiparametric PH mixture cure model.

A.2 Descriptive Statistics for Replication Analysis

Table A.1: Descriptive Statistics for Replication Analysis

Statistic	N	Mean	St. Dev.	Min	Pctl(25)	Pctl(75)	Max
Duration	3931	14.38	13.06	1.00	4.00	21.00	59.00
Civil war Recurrence	3931	0.04	0.19	0.00	0.00	0.00	1.00
Motivation-Decreasing PCJ	4288	0.32	0.53	0	0	1	3
Opportunity-Decreasing PCJ	4288	0.50	0.69	0	0	1	3
Power Sharing	4288	0.06	0.24	0	0	0	1
Military Personnel	4114	5.68	1.06	4.61	4.82	6.16	8.68
GDP/Capita Growth	3978	0.02	0.10	-0.69	-0.01	0.05	1.90
GDP/Capita	3985	21.66	1.00	18.95	20.79	22.40	24.28
Ethnic Conflict	4288	0.39	0.49	0	0	1	1
Democracy	4288	0.21	0.40	0	0	0	1
Conflict Duration	4288	0.78	0.93	0	0	1.4	4
Battle Deaths/Capita	4108	-10.14	2.73	-17.60	-12.04	-8.04	-2.88
Victory	4288	0.49	0.50	0	0	1	1
Peace Agreement	4288	0.10	0.30	0	0	0	1
Number of Rebel Groups	4288	1.30	0.56	1	1	2	4
Peacekeeping Operations	4288	0.10	0.30	0	0	0	1
Post-Cold War	4288	0.56	0.50	0	0	1	1