Not All Intrastate Violence is Civil War: Varieties of Violence and the Perils of Model Dependence in Civil War Research

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Abstract

When is intrastate violence a civil war? Selecting criteria to determine the inclusion and exclusion of cases is one of the most challenging—and contentious—aspects of quantitatively-oriented civil war research. While debate has centered on concerns over selection bias arising from the exclusion of lower magnitude conflicts, we argue that an equally pressing methodological concern—model dependence—can result when conflicts of variable magnitudes are studied uniformly. Major and minor conflicts have different origins and they manifest in different ways. Rather than search for hard and fast thresholds, scholars should instead evaluate their empirical findings across a range of battle death thresholds. Such an approach can provide new leverage when drawing inferences about civil wars, identifying interesting trends in the data that might otherwise be obscured by the strict application of a threshold that is either too high or too low. We illustrate this point with an example drawn from the civil war literature which studies the relationship between drug cultivation and civil war severity.

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1 Introduction

When is intrastate violence a civil war? Most scholars would agree that civil wars involve organized armed conflict between a state and domestic political actors which results in casualties. Determining the appropriate casualty threshold, however, has proven to be a contentious subject in the civil war literature. Thresholds have varied from 1,000 annual deaths per conflict (Small and Singer 1982) to 1,000 cumulative deaths per conflict (Singer and Small 1994), to 25 annual deaths per conflict (Gleditsch et al. 2002). Higher death thresholds help set wars apart from other forms of intrastate violence—for example, riots, crime, terrorism, and coups—but it is well known that they also risk excluding conflicts that may be major wars for smaller countries (Sambanis 2001; 2004). While lower death thresholds guard against this selection bias, they suffer an opposite complication: the threshold is so low that it is not limited to civil wars (Hegre and Sambanis 2006). Faced by these contrary problems, debate over what qualifies as the "correct" threshold has regularly populated the civil war literature (Licklider 1995; Regan 2000; Sambanis 2001; Sarkees, Wayman, and Singer 2003; Sambanis 2004).

While debate has centered on concerns over selection bias arising from the exclusion of lower magnitude conflicts, we argue that an equally pressing methodological concern—model dependence—can result when conflicts of variable magnitudes are studied uniformly. This problem is consequential: as King and Zeng (2007, 184) explain, "[t]he risk with model-dependent inferences is that substantive conclusions are based more on apparently minor modeling choices than on the empirical evidence." Not all intrastate violence is civil war—minor and major conflicts have different origins, and they manifest in different ways. Drawing inferences about the causes, dynamics, and outcomes of intrastate violence across these varieties of violence requires careful case selection, appropriate model specification, and the support of the empirical evidence being analyzed.

Rather than search for hard and fast thresholds, we argue that scholars should instead explore their empirical findings across a range of battle death thresholds. This approach can help to mitigate model dependence by asking "less of the data" and by forcing scholars to hone in on the dynamics operative in particular cases of violence. Using an example drawn from the civil war literature, we show how such an approach can provide new leverage when drawing inferences about conflict, identifying interesting trends in the data that might otherwise be obscured by the strict application of a threshold that is either too high or too low. In particular, we help to clarify the relationship between drug cultivation and armed civil conflict in three ways: first, we show that, despite claims to the contrary, drug cultivation inside the conflict zone has little effect on total battle deaths when excluding low magnitude conflicts; second, that the connection between drug cultivation and decreased combat intensity operates through duration, namely by prolonging civil wars; and third, that this effect of drug cultivation is greater in higher magnitude wars.

This paper proceeds as follows. In section II, we overview the problem of model dependence and illustrate its consequences for civil war researchers by re-analyzing recent

findings concerning the relationship between drug cultivation and the severity of armed civil conflict. In section III, we show how exploring the empirical findings across a range of battle death thresholds clarifies this relationship, but also provides new leverage when drawing inferences about civil wars. Finally, section IV concludes with a discussion of our main findings and how they can improve future studies of civil war.

2 The Perils of Model Dependence

Social scientists studying civil war seek to identify the causal effects of explanatory variables on the onset, duration, severity, or outcomes of armed civil conflicts. To identify causal effects, scholars must estimate differences between factual and counterfactual inferences. However, as King and Zeng (2007, 231) explain,"[w]hen counterfactual questions are posed too far from the data, statistical inference can become highly dependent on seemingly trivial but indefensible modeling assumptions." Put differently, when the causal inferences we make lie far from the observed data we have, they end up being dependent on the particular model we have specified, rather than on empirical evidence. Most scholars seek to guard against this threat by running sensitivity analyses. This usually equates to changing the model to include additional covariates, interaction terms, or fixed effects, and then assessing how much conclusions change. Unfortunately, such analyses are often insufficient tests of the robustness of empirical findings. We must not only show that it is possible to find results consistent with *ex ante* hypotheses, we must also ensure the inferences we draw are supported by the empirical evidence we are analyzing.

2.1 Civil Wars, Drugs, and Counterfactuals: An Illustrative Example

What are the potential consequences of model dependent inference with respect to intrastate conflict? To answer this question, we re-analyze a recent article by Lujala (2009), who investigates the relationship between natural resources and the severity of armed civil conflict. In particular, we examine the article's empirical conclusions regarding the effect of drugs on the total number of combat deaths and combat intensity (the average daily death rate).

Upon analyzing a dataset of 258 civil conflicts with more than 25 battle-related deaths for the period 1946 through 2002, Lujala (2009, 51) finds that "drug cultivation (coca bush, opium poppy, and cannabis) in the conflict zone is associated with fewer combat deaths and lower intensity." The statistical and substantive significance of these associations is large: drug cultivation is shown to decrease both the total number of combat deaths and the intensity of conflict by more than 60 percent, results which are distinguishable from zero at conventional levels. In numbers, this represents a decrease in the yearly death rate from 1500 combat deaths to 600. These results are shown to be robust to the exclusion of outliers, the inclusion of a rich set of additional covariates, and the inclusion of a dummy variable that indicates production status the year before the conflicts start. Taken together, these are striking findings, the magnitudes of which

cut against the conventional wisdom linking drugs to some of the longest, most violent conflicts, from Afghanistan and Burma to Columbia and Mexico (Reyes 1994, Perl 2001, NYT 2012).

What are we to make of these results? We begin our analysis by replicating Lujala's findings (reported in Appendix C). Of the six models included in the table, we focus our analysis on Models 2 and 6, which are Lujala's fully specified models for total combat deaths and combat intensity, respectively, and which feature at the heart of her analysis and discussion.[1]

Assessing the causal effect of drugs on the severity of civil war requires that we identify the counterfactuals of interest by asking the following questions:

1. In civil wars with drug cultivation in the conflict zone, how much more severe would the conflict have been if drugs were not being produced?

2. In civil wars without drug cultivation in the conflict zone, how much less severe would they have been if drugs were being produced?

To assess these counterfactuals formally, we employ the WhatIf software provided by Stoll, King, and Zeng (2006). This tool allows us to assess whether a given counterfactual is an extrapolation or interpolation by identifying whether counterfactual points are within the convex hull of the observed data.[2] Should the analyses fail this test, it will indicate that the author's substantive inferences are sensitive to modeling choices that are not based on empirical evidence.

We perform two counterfactual analyses. The first examines Lujala's findings concerning drug cultivation and total combat deaths. The factual data set has 258 observations on thirteen covariates, as well as our key causal variable, DRUGP (a dummy variable indicating whether there is drug cultivation in the conflict zone). A total of 14 observations include missing values; following Lujala, we list-wise delete these observations. We construct a counterfactual dataset that is identical to the factual dataset, save for the replacement of DRUGP with 1-DRUGP. The second counterfactual analysis examines Lujala's findings concerning drug cultivation and combat intensity. The factual dataset now adds a fourteenth covariate, a dummy indicator for conflicts with durations of 10 days or less. Again, 14 observations include missing values, and we follow Lujala by list-wise deleting them. The counterfactual dataset is again identical to the factual dataset, save for the replacement of DRUGP with 1-DRUGP.[3]

[1] We have repeated this analysis with various specifications including removing potentially unrelated covariates as well as testing potential interactive effects. These changes do not substantially alter the author's results. Further, in each of the models employed by the author, there may be post-treatment bias driven by a potential relationship between drug cultivation and state and rebel strength (acknowledged in Lujala 2009, 62). When we carry out this analysis by excluding these variables, we do not observe notable differences in the estimated impact of drug cultivation.

[2] The convex hull of a set of points is defined as the smallest convex set that contains them. For elaboration, see King and Zeng (2006) and King and Zeng (2007).

[3] Note that we have also carried out these analyses using multiple imputation to fill-in values across five datasets for the missing observations. This procedure has no impact on the author's results.

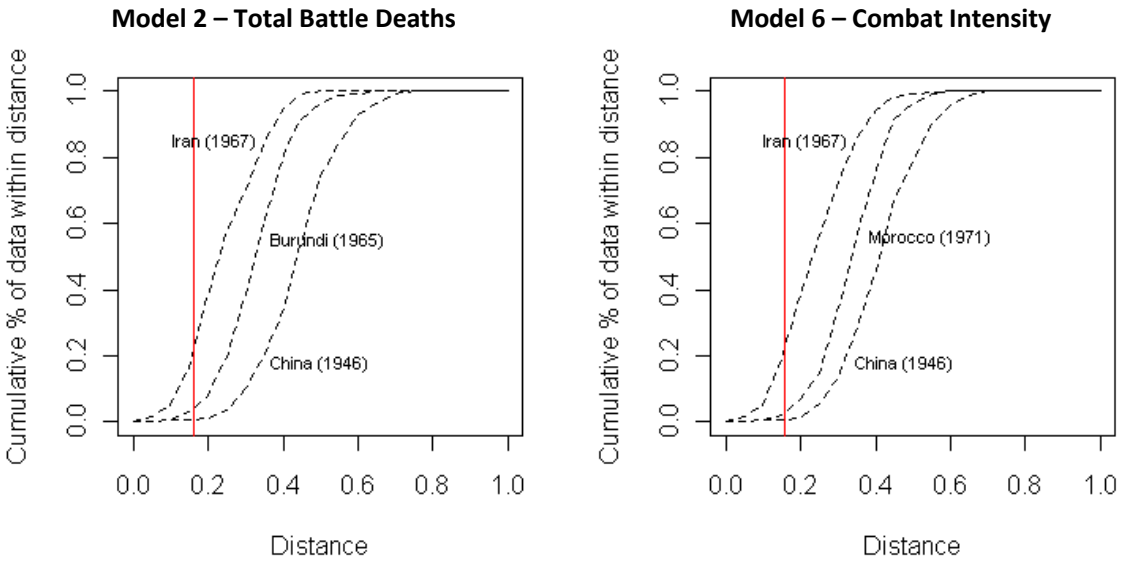


Figure 1: Cumulative Frequencies of Distances to Counterfactual

Results of the convex hull test indicate that in both analyses, and for every element analyzed, there are no corresponding counterfactuals in the convex hull of the observed data. In other words, every counterfactual in both datasets is an extrapolation from the empirical support of the data. This strongly suggests that these counterfactuals suffer from serious model dependence, thereby holding little empirical content for causal inference.

Next, we calculate Gower distances of each counterfactual from the data to find which counterfactuals are at least near the convex hull. To define observations as "sufficiently close" to their respective counterfactuals, we follow the advice of King and Zeng (2006, 138), who suggest researchers use the fraction of observations in the data with pairwise Gower distances less than the geometric variability (GV) of X (roughly, the average distance among all pairs of observations in the data). Our results for the first counterfactual analysis (concerning total battle deaths) show that the GV of Lujala's observed data is 0.161, and that only a fraction—3.65 percent—of the observed data meets this threshold. Likewise, results for the second counterfactual analysis (concerning combat intensity) show that the GV of Lujala's observed data is 0.158, and that only 4.19 percent of the observed data meets this threshold.

Figure 1 plots the cumulative frequency distributions of the minimum, median, and maximum distances for both counterfactual analyses, with the vertical line in these plots representing their respective GV values. The figure shows that in both counterfactual analyses, the counterfactual with the minimum percent of data within the GV value is the 1946 civil war in China, with 0.004 percent. At the medians, only 2.87 percent of the data is within the GV values for counterfactuals on Burundi (1965) and Morocco (1971). The counterfactual with the maximum percent of data within the GV value for both counterfactual analyses is the 1967 civil war in Iran, but even then only 20 percent

of the data is within this distance.

We take this evidence to indicate that the counterfactuals we are exploring are not just extrapolations—they are not even close to the convex hull. Again, this is strong evidence that the findings in Lujala (2009) result from model specifications, rather than empirical evidence. We investigate the source of this model dependence in the next section.

2.2 Different Violence, Different Characteristics: Searching for Common Support

Intrastate violence can take many forms – demonstrations and riots, attacks against other groups, coups, purges, or civil wars. While related, these "varieties of violence" stem from different underlying causes, and emerge under varying conditions. Studying them in tandem may be appropriate for some research questions, but may often be inappropriate for investigating the causes, dynamics, and consequences of civil war.

Consider the dataset analyzed herein. Included is a conflict in Ethiopia (1996), which lasted 1,243 days and killed 50 people, resulting in a daily death rate of 0.04 people per day. This conflict is coded as having drug cultivation in the conflict zone. Also included in the dataset is a conflict in China (1946), which lasted 1,445 days, killing an estimated 1.2 million people at a rate of over 830 people per day. This conflict is coded as one without drug cultivation in the conflict zone. Before raising concerns about regional and temporal variation between these two conflicts, we might first ask how appropriate it is to compare these very different varieties of violence. The all-out war in mainland China from 1946-1950 was fought between ideological foes and waged by conventional armies fielding millions of men. By contrast, the violence waged by the al-Itahad al-Islami militant group in the Jigjiga Zone of Ethiopia from 1996-2000 took the form of minor border raids, staged from disputed areas between Somalia and Ethiopia. Is it appropriate to estimate the effect of drugs on the severity of these two conflicts?

Causal inference requires equality in the support of X in the treated and control groups (i.e. $Support[X|T = 1] = Support[X|T = 0]$). Without this equality, we cannot rule out the possibility that for some values of covariates (whether observed or not) there are some treated units for which we cannot find any comparable control units. Thus, if our dataset included an observation that matched the case of Ethiopia as closely as possible, differing only on the explanatory variable of interest (i.e. drug cultivation in the conflict zone), the disparity between the Ethiopian and Chinese cases would pose little threat to our ability to undertake causal inference. In the absence of this "common support," however, estimates of causal effects become problematic.

To make this point concrete, we preprocess Lujala's data using matching techniques in an effort to ameliorate model dependence as far as possible. Of the numerous techniques available, we find the genetic matching algorithm described by Diamond and Sekhon (2005) and implemented by Ho, Imai, King, and Stuart (2007) in the MatchIt software package to provide the lowest covariate imbalance after matching.[4]

[4]In our efforts to maximize covariate balance, we employed a number of matching algorithms including Mahalanobis Distance, Propensity Scores, Coarsened Exact Matching, and Optimal.

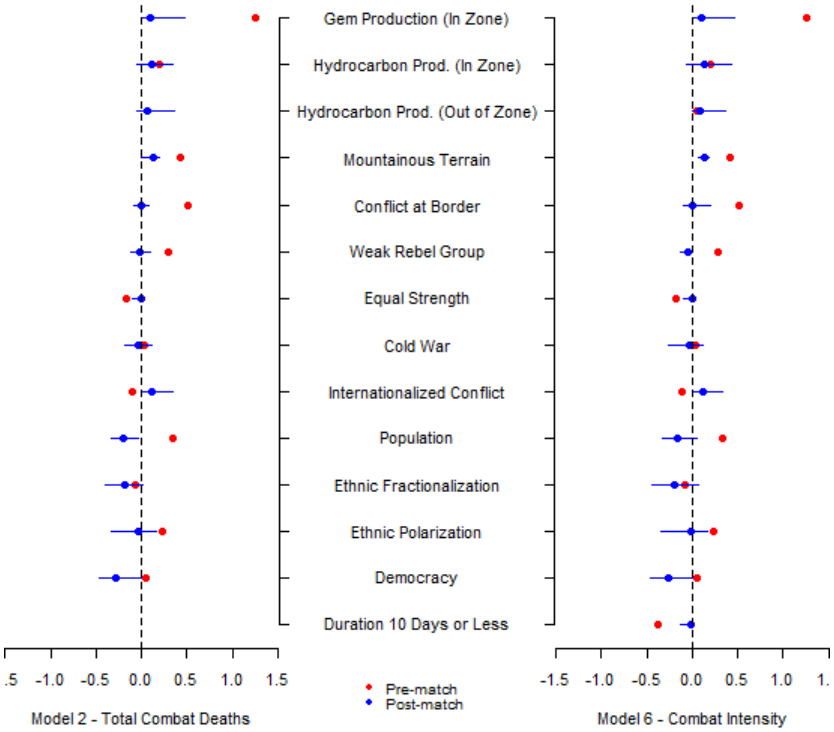


Figure 2: Mean Standardized Imbalance from 1,000 Genetic Matches (95% Confidence intervals presented for the post-match standardized imbalance. Note that the pre-match standardized imbalance for hydrocarbon production out of the conflict zone in both Models 2 and 6 is obscured by the post-match standardized imbalance value)

Given the absence of counterfactuals inside the convex hull, matching techniques can only improve covariate balance between treated and controls to a limited degree. Indeed, we find that matching results are variable, with the post-matching balance shifting between iterations. To investigate the extent of this matching variability, we pre-process the data with genetic matching 1,000 times. We plot the mean standardized imbalance from each of these runs in Figure 2 (covariate imbalance statistics are reported in Appendix A). The 95% confidence intervals indicate that a number of covariates—for example, Gem Production, Democracy, and Ethnic Polarization—confound the matching algorithm. Taken as a whole, however, the balance achieved post-matching is always superior to that of the original datasets. As such, we briefly investigate the impact of this data pre-processing on the author's results.

Recognizing the volatility of the matching procedure, rather than drawing conclusions from individual matching iterations, we study the influence of pre-processing the data by plotting the posterior distributions of the exponentiated drug cultivation coefficients and T-statistics from 1,000 matched datasets. The results are depicted in red in

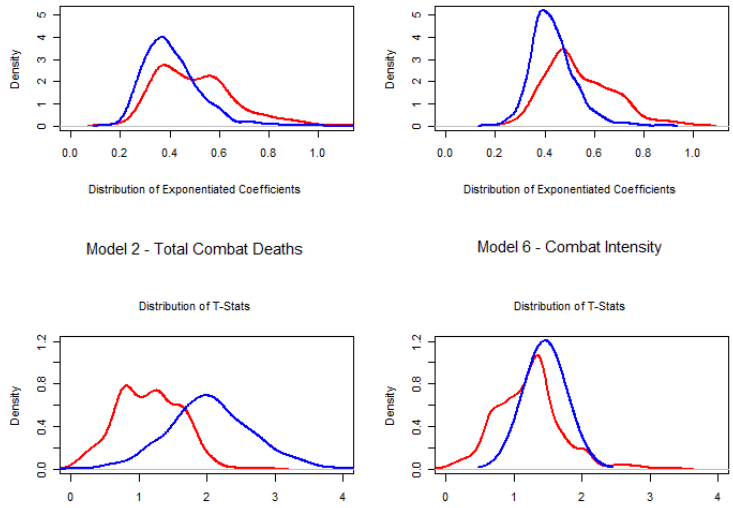


Figure 3: Distribution of T-Statistics and Exponentiated Coefficients for 1,000 Simulated Matched Datasets (in red) and 1,000 Datasets From Randomly Dropping Observations Parallel to the Number Dropped by Matching (in blue)

Figure 3.[5] The distributions show a slight attenuation of the magnitude of the author's exponentiated coefficients for drug cultivation. More importantly, for both Models 2 and 6, the distributions show increasing uncertainty, with the mean T-statistics on the effect of drug cultivation falling to 1.14 and 1.22 in Models 2 and 6, respectively.

Since this result could arise from a loss of statistical efficiency from dropping observations, we simulate 1,000 datasets by randomly dropping the number of observations excluded by genetic matching. We plot the posterior distribution of these estimated exponentiated coefficients and T-statistics in blue in Figure 3. Comparing the randomly dropped and matched distributions, we note a clear difference with respect to Model 2, where the mean of the randomly dropped T-statistics is 2.05. This suggests that the increased uncertainty we identified with respect to the role of drug cultivation can be at least partially attributed to pre-processing the data. We are unable to draw similar conclusions with respect to Model 6 as the distribution of T-statistics from random and matched are comparable. Nonetheless, this exercise illustrates the importance of common support and the threat of model dependence when estimating causal effects.

3 Re-Evaluating the Relationship between Drug Cultivation and Intrastate Violence

The inability to achieve consistent matches, as well as the sensitivity of the author's results to even non-optimal data pre-processing, underscores the pitfalls of model de-

[5]While we recognize that exponentiated coefficients are not an ideal quantity of interest (and thus simulate first differences in expected battle deaths and conflict intensity below), we use Lujala's metrics to provide a simple test of the differences between her findings and those uncovered by matching.

pendence. Indeed, given that the underlying characteristics of violence vary substantially as a function of many of the variables included in the matching analysis, it is probable that inferences drawn uniformly across different magnitudes of violence will be highly dependent on modeling procedures which extrapolate across dissimilar observations—such as Ethiopia (1996) and China (1946). We argue that more flexible approaches which recognize the potential for variable causal processes across different varieties of violence can help reduce reliance on modeling decisions by asking "less" of the data. In addition, such analysis can help to uncover heretofore overlooked conflict dynamics operative at varying magnitudes of conflict. In this section, we illustrate the utility of such an approach by re-evaluating the author's findings across different battle death thresholds.

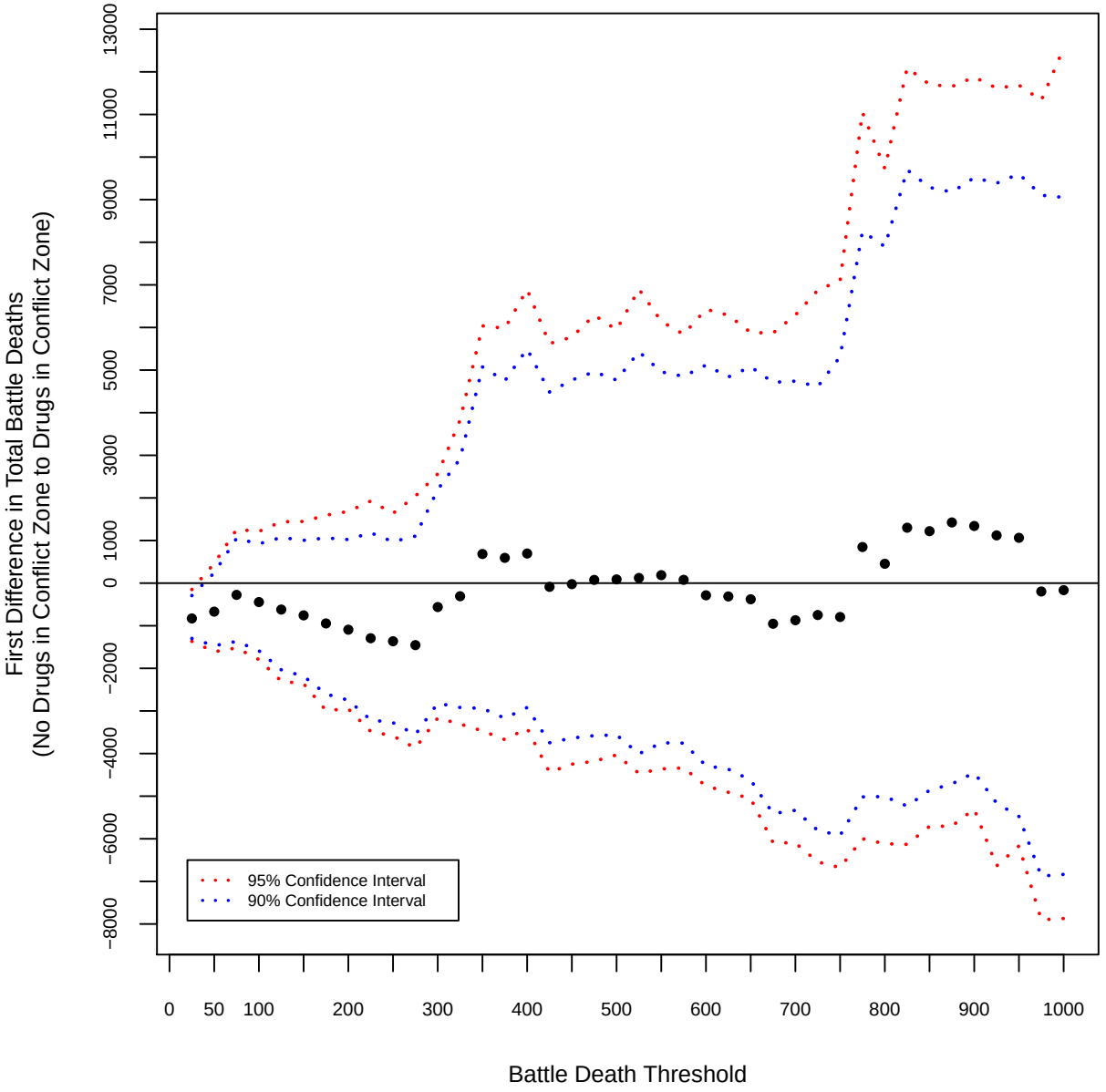
3.1 Total Conflict Battle Deaths

Figure 4 expands on the author's analysis of drug cultivation and total battle deaths by investigating the effect of iteratively increasing the battle death threshold from 25 deaths (adopted by the author) to 1,000 deaths. Rather than showing exponentiated coefficients, we present the simulated change in the number of battle deaths associated with a shift from conflicts without drug cultivation to those with drug cultivation.[6]

We uncover the sensitivity of the author's finding that drug cultivation reduces total battle deaths. In particular, we find that the relationship only holds for small wars with less than 50 total battle deaths. At thresholds greater than 50 total battle deaths, the 90% and 95% confidence intervals cross zero. More importantly, these confidence intervals expand rapidly as the battle death threshold increases, indicating rising uncertainty. What is more, the estimated impact of drug cultivation becomes unstable, first decreasing in magnitude, and then even switching signs to show a positive association. In short, taking into account potential dissimilarities between conflicts of varying sizes shows that the relationship uncovered by the author does not hold across many of the conflicts in the dataset.

One potential concern with this finding is that the statistical weakness and substantive variability we identify are simply a function of having deleted observations when excluding smaller conflicts. However, as shown in the table accompanying Figure 4, this scenario seems unlikely, as the author's result is attenuated even when shifting to a threshold of 50 battle deaths, which only excludes 22 conflicts (of which only four had drug cultivation). Moreover, even at the 1,000 death threshold, nearly half of the original observations remain, 17 of which are conflicts with drug cultivation. Note also that the ratio of conflicts with drug cultivation to those without stays consistent at between 11-13% across all thresholds.

[6] We estimate these first differences by first simulating expected values of $log(TotalBattleDeaths)$ when drug cultivation is set to "0" and "1" and then subtracting the exponentiated expected value of conflicts with drug cultivation from those without. Note that while we set all other explanatory variables at their means, this choice is irrelevant in a linear model where the first difference is based on the coefficient on the treatment, in this case, drug cultivation.



Battle Death Threshold	Observations	Drug Wars
25	244	34
50	222	30
75	205	26
100	199	26
125	191	25
150	187	24
175	181	24
200	178	24
225	171	24
250	170	24
275	165	23
300	162	21
325	152	20
350	149	19
375	148	19
425	145	19
525	141	19
575	140	19
600	139	19
625	137	19
675	130	19
725	129	19
750	128	19
775	127	18
800	126	18
825	124	17
925	121	17
950	120	17
975	117	17

Figure 4: First difference in total battle deaths (from no drug cultivation to drug cultivation) at varying battle death thresholds. The accompanying table lists the thresholds which resulted in more observations being removed.

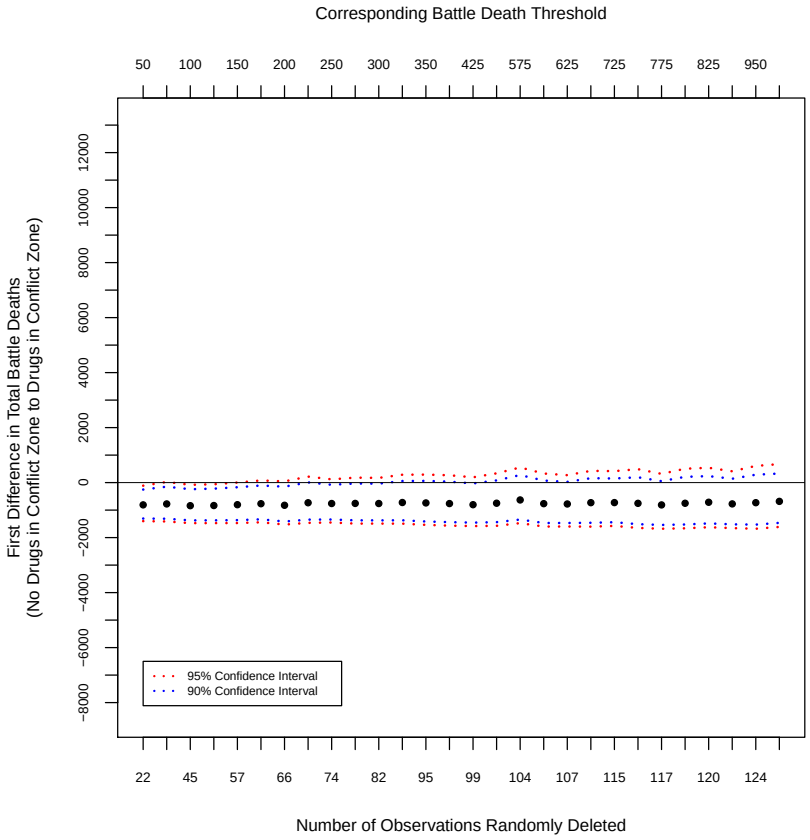


Figure 5: Mean first difference in battle deaths (from no drug cultivation to drug cultivation) when randomly deleting observations to accord with the number of observations excluded by each battle death threshold. Each point estimate represents the mean first difference from 100 randomly simulated datasets.

Nonetheless, we address this concern more rigorously by simulating the effect of dropping observations on the first difference in total battle deaths through randomly deleting the equivalent number of observations excluded by each threshold 100 times.[7] Figure 5 presents the mean first difference estimates from each of the 100 randomly simulated datasets. The pattern in the figure confirms that the relationship between drug cultivation and total deaths at different battle death thresholds is not driven by a loss of statistical efficiency related to dropping observations. Indeed, the theorized first difference Lujala estimates at the 25 battle death threshold remains stable at approximately -800 deaths, while the 90% confidence intervals barely cross zero when over 100 observations are deleted. Though this represents a slight loss of statistical efficiency, it is not comparable to the level of uncertainty depicted in Figure 4.

[7]Note that we randomly simulate 100 datasets for each unique number of remaining observations. As such, if the shift to a higher battle death threshold does not exclude additional observations, we only simulate that number of missing observations once. The list of unique numbers of remaining observations is in the second column of the table accompanying Figure 4.

3.2 Conflict Intensity

Just as we uncovered new patterns with respect to total battle deaths, so too do we find interesting results with respect to conflict intensity. The author argues that there is an added effect of drug cultivation on conflict intensity beyond that of duration (2009, 63-64). But consider the author's measure of intensity:

$$ConflictIntensity = \frac{TotalBattleDeaths}{ConflictDuration}$$

We have shown that there is no relationship between drugs and battle deaths (the numerator) for conflicts with 50 total battle deaths or more. Thus, we would expect drug cultivation to have little influence on intensity at higher battle death thresholds except through increasing conflict duration (the denominator). Furthermore, if duration is indeed the channel through which drug cultivation influences conflict intensity, we would expect the relationship between drug cultivation and intensity to diminish once controlling for duration at higher battle death thresholds.

We turn first to the relationship of drug cultivation and intensity without controlling for duration. Figure 6 plots the simulated first differences, at different battle death thresholds, in deaths per day when shifting from a conflict with no drug cultivation to one in which there is drug cultivation. We find that the association between drug cultivation and conflict intensity identified by the author holds at different battle death thresholds. Moreover, the figure suggests that the pattern may be even more extreme than Lujala identifies: at the 700 battle death threshold, moving from a conflict without drug cultivation to one with drug cultivation is associated with nearly six fewer deaths per day, as compared to the two fewer deaths per day reported by the author.

However, in Figure 7, we present evidence that this relationship is driven by duration. Indeed, once we control for duration, both the 90% and 95% confidence intervals cross zero and remain above zero after passing the 300 battle death threshold. Moreover, the distribution of point estimates and confidence intervals differs markedly between the models with and without a duration control.

To illustrate that these findings are not due to the statistical effects of dropping observations, we once again randomly simulate 100 datasets corresponding to the number of observations dropped at each battle death threshold and calculate mean first differences in drug cultivation for each unique threshold of dropped observations. These results, reported in Appendix B, confirm that the findings with respect to changing battle death thresholds and conflict intensity are due to the change in threshold, not a decrease in statistical efficiency. In sum, we again find that taking into account the battle death threshold, and thus not relying on the model to estimate effects across different magnitude conflicts, reveals limitations within the author's findings.

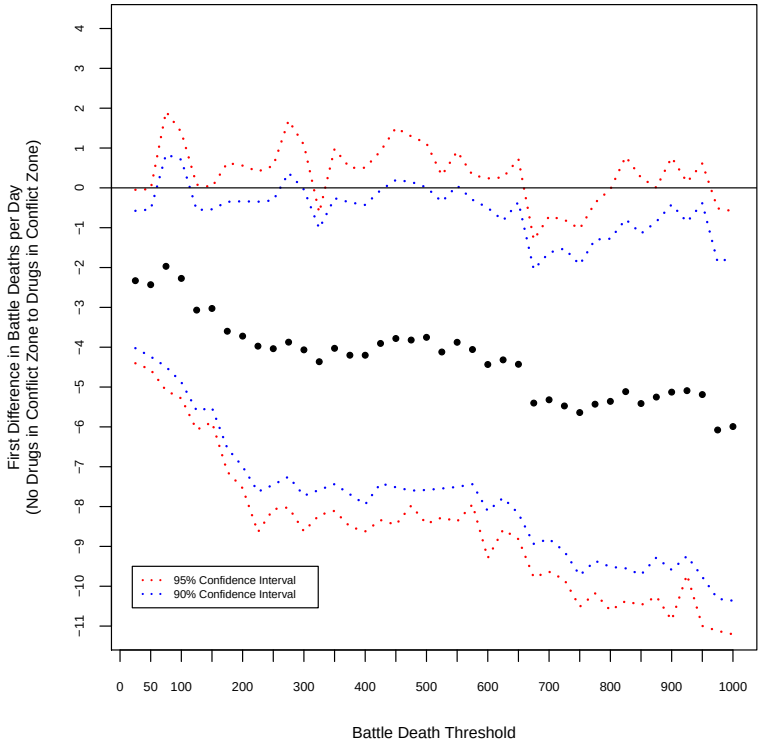


Figure 6: First difference of drug cultivation on conflict intensity at varying battle death thresholds (No Duration Control)

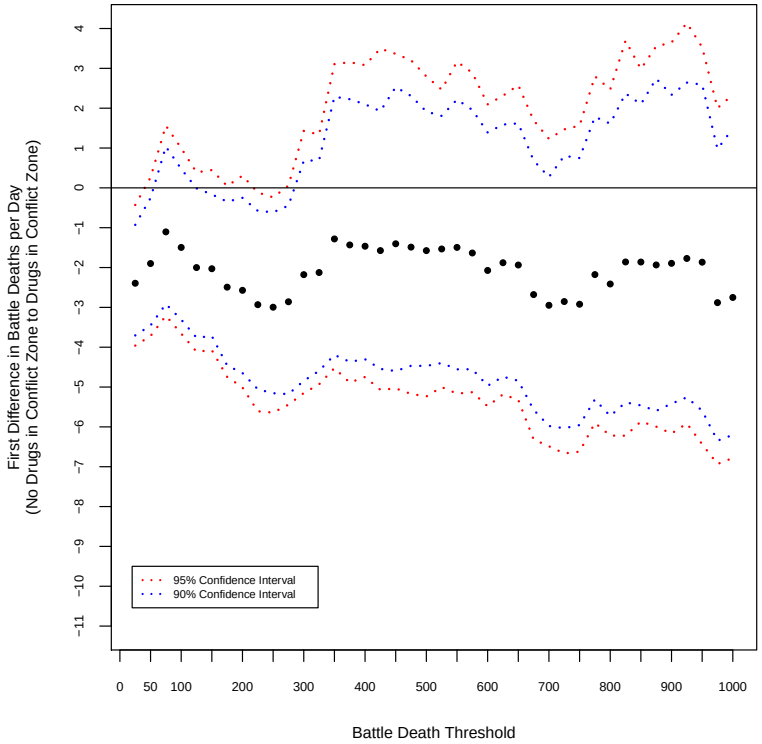


Figure 7: First difference of drug cultivation on conflict intensity at varying battle death thresholds (Including Duration Control)

3.3 Connecting the Dots: Drug Cultivation and Conflict Duration

Given the above findings, we theorize a link between the results reported in Lujala (2009) and a broader literature which finds that drug cultivation increases the duration of conflict. In particular, Fearon (2004), as well as Ross (2003, 2004a, 2004b), argue that drug cultivation provides incentives to lengthen conflict as it makes available potential resources for insurgents to finance themselves and thus keep fighting. We test this hypothesis with a Weibull duration model,[8] employing the set of covariates analysed in Lujala's fully saturated model 8. This model includes a dummy for conflicts over territory and an interaction of territorial conflicts and hydrocarbon production in the conflict zone.[9] We include these additional covariates as secessionist conflicts are theorized to be associated with longer wars. The logic here is threefold: first, central governments may be driven to forcefully retain secessionist regions to ensure access to fuel or mineral resources located in that territory; second, central governments are likely to react forcefully to secessionist threats due to fears that secession will set an unwanted precedent for other groups calling for autonomy from the state; and third, these conflicts tend to last longer than other civil wars because self-rule and control over a "homeland" territory are perceived by rebel groups to be non-divisible goods (Licklider 1993; Balch-Lindsay and Enterline 2000; Goddard 2006; Wallensteen et al. 2002; Themner and Wallensteen 2011).

The results from simulating the association of drug cultivation with the first difference in expected conflict days using the Weibull model are presented in Figure 8. They show an upward trending pattern as the battle death threshold increases, indicating that in larger magnitude conflicts, drug cultivation is associated with longer conflicts. For example, after the 500 battle death threshold, conflicts with drug cultivation are nearly 2,000 days longer. Though the confidence intervals cross zero, they become comparatively tighter as the battle death threshold increases. As such, we find tentative evidence supporting the literature's conclusions concerning the connection between drug cultivation and conflict duration. Indeed, when including interactions between secessionist conflict and drug cultivation as well as gem production, an interactive relationship suggested by Ross (2004a), the substantive results increase and the confidence intervals do not cross zero above the 50 battle death threshold. This result provides additional traction in explaining our finding that drug cultivation in higher battle death wars is associated with less intense conflicts. In short, when high death counts are spread over a longer time period, they are less intense. Critically, this relationship is only apparent when examining the relationship between drug cultivation and duration at different battle death thresholds.

[8] We use the Zelig software package to complete this regression and to estimate first differences in expected duration. Imai, Kosuke, Gary King, and Olivia Lau. "Zelig: Everyone's Statistical Software" (2006). http://gking.harvard.edu/zelig

[9] Note that though the substantive results don't change with the use of the author's other models (i.e. Lujala's Models 2 and 6), the confidence intervals are slightly further from zero.

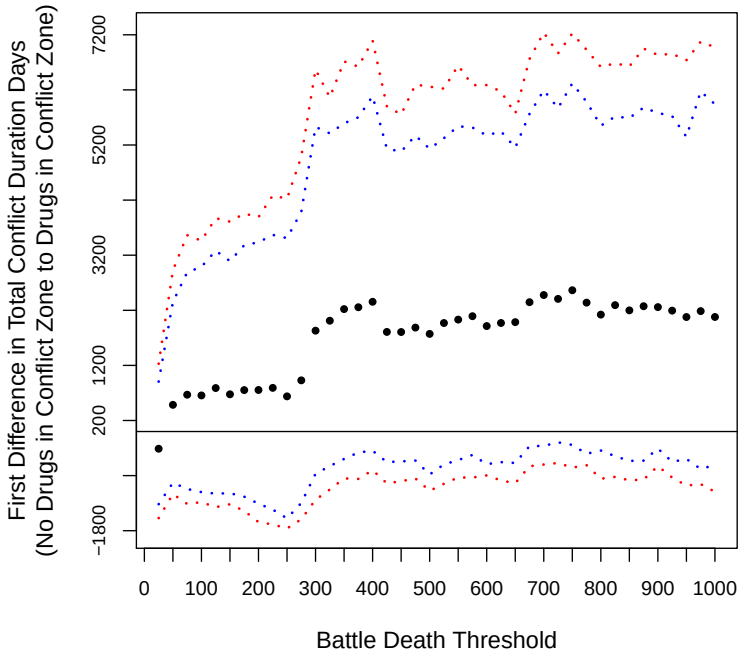


Figure 8: First difference in conflict duration days when moving from conflicts with drug cultivation to those without across varying battle death thresholds

4 Conclusion

As King and Zheng (2007, 208) observe, the study of many interesting questions is plagued by non-existent counterfactuals. Not surprisingly, the study of civil war is often beset by such concerns due at least in part to the fact that major and minor conflicts have different origins and manifest in different ways. As illustrated by our analysis of Lujala (2009), a lack of common support can lead to model dependence whereby researchers extrapolate results beyond the empirical evidence they marshal. While the stark differences between China in 1946 and Ethiopia in 1996 have served as a motivating example in this paper, many studies of civil war do not acknowledge the challenges posed by drawing inferences from fundamentally dissimilar units. In this article, we illustrate the impact of such model dependence by studying the relationship of drug cultivation and conflict severity at different battle death thresholds. We clarify this relationship in three ways: first, despite claims to the contrary, drug cultivation inside the conflict zone has little effect on total battle deaths when excluding low magnitude conflicts; second, that the connection between drug cultivation and decreased combat intensity operates through duration, namely by prolonging civil wars; and third, that this effect of drug cultivation is greater in higher magnitude wars. We illustrate that rather than searching for hard and fast thresholds of violence—be they low thresholds of 25 deaths

or high thresholds of over 1,000 deaths—researchers are better served by evaluating their empirical findings across a range of battle death thresholds. Such analysis helps mitigate the dangers of model dependence by "asking less of the data" and forcing more focused analysis of the dynamics operative in particular cases of conflict.

Importantly, the study of varying battle death thresholds is not, and should not be, the only way to account for the differences in conflict characteristics observed across cases of intrastate violence. Indeed, as this paper emphasizes, potential theoretical and empirical boundaries need not engender debate about the "right" and "wrong" levels of analysis. Instead, researchers should embrace these boundaries in their data, studying the ways they inform interesting questions about conflict and the varieties of violence.

A Matching Results

Below, we show the imbalance statistics for Models 2 and 6. The statistics shown for post-balance are the mean values from 1,000 simulations of genetic matching.

Table 1: Average Balance Results for 1,000 Simulations of Genetic Matching for Model 2 Data

	Pre Dif. Means	Post Dif. Means	Pre L1	Post L1
Gem Production (In Zone)	-0.4756	0.1211	0.4756	0.1398
Hydrocarbon Prod. (In Zone)	-0.0919	0.1029	0.0919	0.1097
Hydrocarbon Prod. (Out Zone)	-0.0294	0.0736	0.0294	0.0826
Mountainous Terrain	-2.8505	0.7351	0.0143	0.0047
Conflict at Border	-0.2482	0.0624	0.2482	0.0684
Weak Rebel Group	-0.1165	0.0417	0.1165	0.0479
Equal Strength	0.0555	-0.0373	0.0555	0.0416
Cold War	-0.0176	-0.0209	0.0176	0.0471
Internationalized Conflict	0.0356	-0.0077	0.0356	0.0459
Population	-0.5712	0.1061	0.0868	0.0640
Ethnic Fractionalization	0.0214	-0.0311	0.1022	0.0667
Ethnic Polarization	-0.0880	-0.0175	0.0880	0.0453
Democracy	-0.0210	-0.0126	0.0210	0.0302

Table 2: Average Balance Results for 1,000 Simulations of Genetic Matching for Model 6 Data

	Pre Dif. Means	Post Dif. Means	Pre L1	Post L1
Gem Production (In Zone)	-0.4756	0.1434	0.4756	0.1452
Hydrocarbon Prod. (In Zone)	-0.0919	0.1114	0.0919	0.1128
Hydrocarbon Prod. (Out Zone)	-0.0294	0.0813	0.0294	0.0869
Mountainous Terrain	-2.8505	0.8119	0.0143	0.0016
Conflict at Border	-0.2482	0.0675	0.2482	0.0686
Weak Rebel Group	-0.1165	0.0349	0.1165	0.0369
Equal Strength	0.0555	-0.0395	0.0555	0.0409
Cold War	-0.0176	-0.0112	0.0176	0.0475
Internationalized Conflict	0.0356	0.0012	0.0356	0.0467
Population	-0.5712	0.1195	0.0868	0.0668
Ethnic Fractionalization	0.0214	-0.0469	0.1022	0.0563
Ethnic Polarization	-0.0880	-0.0168	0.0880	0.0479
Democracy	-0.0210	-0.0040	0.0210	0.0342
Duration 10 Days or Less	0.1468	-0.0221	0.1468	0.0226

B Replication of Threshold Figures with Randomly Dropped Observations

As illustrated in Figure 5, the changes observed when changing the battle death threshold are not due to losses in statistical efficiency due to dropped observations. In this section, we present similar plots for all of the figures used during our analysis. In each plot, the X axis represents the number of observations dropped from the original dataset. For each of these "drop thresholds," we simulate the process 100 times.

B.1 Replication of Conflict Intensity Figures using Randomly Dropped Observations

The replicated figure for the conflict intensity model with and without a duration control are below. Neither of these figures resemble the patterns seen in Figures 6 and 7.

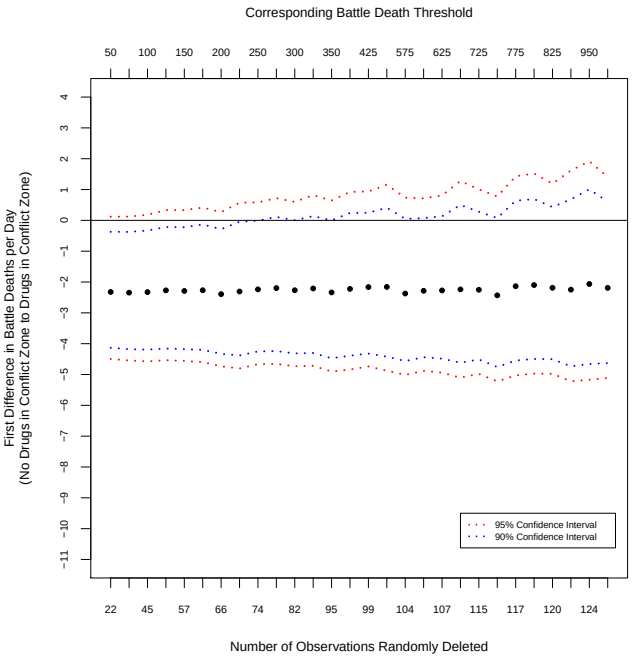


Figure 9: First difference in deaths per day (no drug cultivation to drug cultivation) when randomly deleting observations to accord with the number of observations excluded by each battle death threshold. Each unique set of dropped observations is simulated 100 times (No Duration Control).

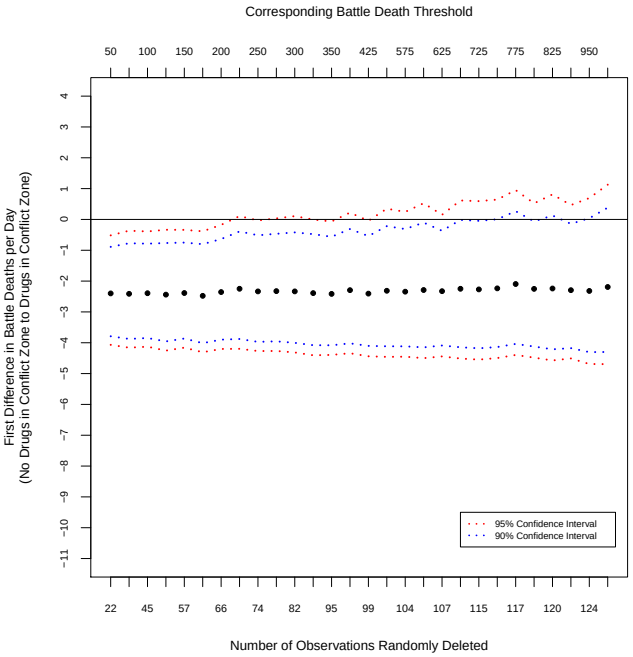


Figure 10: First difference in deaths per day (no drug cultivation to drug cultivation) when randomly deleting observations to accord with the number of observations excluded by each battle death threshold. Each unique set of dropped observations is simulated 100 times (Duration Control).

B.2 Replication of Conflict Duration Model using Randomly Dropped Observations

The replicated Weibull model does not exhibit the pattern seen in Figure 8.

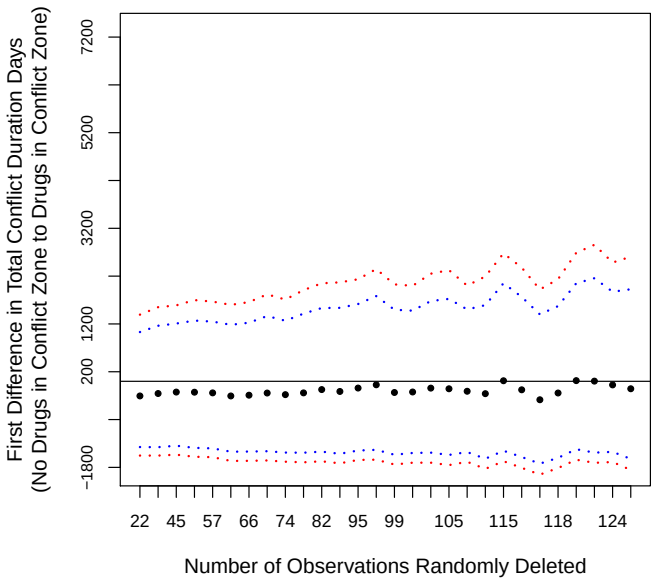


Figure 11: First difference in conflict days (no drug cultivation to drug cultivation) when randomly deleting observations to accord with the number of observations excluded by each battle death threshold. Each unique set of dropped observations is simulated 100 times.

C Replication of Paivi Lujala, "Deadly Combat over Natural Resources: Gems, Petroleum, Drugs, and the Severity of Armed Civil Conflict" from *Journal of Conflict Resolution* 2009 53:50

We have been able to replicate each of the results from *Deadly Combat over Natural Resources: Gems, Petroleum, Drugs, and the Severity of Armed Civil Conflict*. In the following two sections, we go through each of the major tables, indicating any concerns or difficulties we had in the replication process.

C.1 Replicating Table 3

Table 3 provides the results from Lujala's Table 3 (p. 61). Note that the coefficients are exponentiated and the t-values for the robust standard errors are in parentheses. As such, the coefficients can be read in multiplicative terms: movement to a conflict zone with drug production is associated with a 60% decrease in total battle deaths (i.e. it is 0.6 less than one).

C.2 Replicating Table 4

Table 4 provides the results from Lujala's Table 4 (p. 64). We have shown all of the additional coefficient values as well instead of omitting them as done in the original. Note that the T-statistics are below the estimates, not to the side.

Table 3: Replication of Lujala Table 3: OLS Estimates for Total Combat Deaths and Daily Death Rate (Intensity) of Armed Civil Conflict, 1946-2002

	Total Combat Deaths			Intensity		
	1	2	3	4	5	6
Gem production,	2.565	2.720	2.754	1.761	1.163	1.592
conflict zone	(2.05)	(2.51)	(2.34)	(1.46)	(0.33)	(1.13)
Drug cultivation	0.445	0.388	0.693	0.398	0.407	0.422
conflict zone	(1.86)	(2.39)	(0.78)	(2.50)	(2.03)	(2.21)
Hydrocarbon production,	3.007	2.558	3.679	1.845	1.351	1.570
conflict zone	(3.75)	(3.41)	(4.41)	(2.53)	(0.92)	(1.85)
Hydrocarbon production,	0.583	0.481	0.582	0.561	0.650	0.586
outside conflict zone	(1.79)	(2.55)	(1.91)	(2.06)	(1.18)	(1.92)
Mountainous terrain (log),	1.052	1.061		1.055	1.049	1.060
conflict zone	(2.30)	(2.98)		(2.85)	(1.82)	(3.46)
Conflict at border,	3.062	2.470		1.002	0.424	0.886
conflict level	(3.31)	(2.75)		(0.01)	(2.55)	(0.43)
Weak rebel group		1.262	1.560	0.419	0.146	0.346
		(0.37)	(0.69)	(2.05)	(3.84)	(2.29)
Equal strength		4.410	3.756	1.669	0.661	1.567
		(2.11)	(1.75)	(1.07)	(0.70)	(0.86)
Cold War		3.885		3.521	3.206	2.690
		(4.08)		(4.31)	(3.40)	(3.43)
Internationalized Conflict		10.702	6.920	4.096	1.639	2.870
		(5.72)	(3.96)	(3.98)	(1.43)	(3.49)
Population size (log)		1.590		1.286	1.051	1.254
		(5.46)		(2.57)	(0.37)	(2.18)
Ethnic fractionalization		0.259		0.213	0.176	0.191
		(2.43)		(3.07)	(2.51)	(3.21)
Ethnic polarization		0.454		0.392	0.341	0.332
		(2.00)		(2.72)	(1.96)	(2.87)
Democracy		0.481	0.691	0.401	0.337	0.292
		(2.07)	(0.87)	(3.17)	(3.15)	(4.52)
Duration (log)				1.668		
				(10.78)		
10 days or less dummy						37.601
						3.63
N	258	244	247	244	244	244
F	8.59	23.23	8.99	31.48	9.67	24.50
R^2	0.15	0.39	0.17	0.58	0.37	0.58
adj. R^2	0.13	0.35	0.14	0.55	0.33	0.55

Exponentiated coefficients presented. Absolute t-values, adjusted over countries, in parentheses. Dependent variables are log-transformed.

Table 4: Replication of Lujala Table 4: OLS Estimates for Total Combat Deaths and Daily Death Rate of Armed Civil Conflict, 1946-2002: Interaction Effects

	Combat Deaths	Intensity
	7	8
Gem production,	3.014	1.606
conflict zone	(2.640)	(1.120)
Drug cultivation,	0.397	0.448
conflict zone	(2.380)	(2.040)
Hydrocarbon production,	1.804	0.942
conflict zone	(1.470)	(0.200)
Hydrocarbon production,	0.465	0.563
outside conflict zone	(2.830)	(2.160)
Mountainous terrain (log),	1.058	1.057
conflict zone,	(2.840)	(3.170)
Conflict at border,	2.377	0.918
conflict level	(2.550)	(0.310)
Weak rebel group	1.240	0.365
	(0.350)	(2.120)
Equal Strength	4.201	1.639
	(2.100)	(0.950)
Cold War	3.837	2.555
	(4.130)	(3.510)
Internationalized Conflict	11.344	3.137
	(5.700)	(3.670)
Population size (log)	1.596	1.348
	(4.510)	(2.450)
Ethnic fractionalization	0.237	0.176
	(2.600)	(3.300)
Ethnic polarization	0.464	0.364
	(1.930)	(2.760)
Democracy	0.470	0.296
	(2.080)	(4.200)
Duration (log)	0.932	0.565
	(0.150)	(1.310)
Hydrocarbon x Territorial Conflict	2.269	2.847
	(1.350)	(1.900)
Dummy for duration		36.360
		(11.540)
F: Joint significance test	6.01	2.59
F	20.56	24.85
N	244	244

Absolute t-values, adjusted over countries, in parentheses. Models 7 and 8 include the same covariates as models 2 and 6 in table 3, respectively

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