Do Girls Pay the Price of Civil War? Violence and Infant Mortality in Congo*

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Abstract

This paper documents the impact of civil wars in the Democratic Republic of Congo on infant mortality between 1997 and 2004. It adopts an instrumental variable approach to correct for the non-random timing and location of conflict and its liekely correlation to our dependent variable. Strong and robust evidence, including mother fixed effects regressions, shows that conflict significantly increases girl mortality. It also examines the mechanisms explaining this phenomenon, with a focus on disentangling the behavioral from the biological factors. The analysis suggests that gender imbalances in infant mortality are driven by the selection induced by a higher vulnerability of boys in utero rather than by gender discrimination.

Keywords: civil war, infant mortality, gender discrimination.

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1 Introduction

The impact of violence on the demography of a society can substantially increase the overall costs of a conflict, and heavily affect the time and the nature of the recovery process (Ghobarah, Huth, and Russett 2003; Chen, Loayza, and Reynal-Querol 2008). A solid knowledge of the effects of war on the most fragile section of the population therefore represents a necessary condition for devising the proper responses to protect the next generations in conflict-prone environments, given the persistence over the life cycle of the detrimental impact of shocks experienced in early life (Aguero and Deolalikar 2012; Akresh et al. 2012; Dominguez and Barre 2013).

A variety of factors may have a negative impact on infants' health. Malnutrition, resulting from the contraction of the internal supply of food and a partial collapse of trade in the regions in which violence unravels, for instance, worsens the general health status of the affected population (Alderman, Hoddinott, and Kinsey 2006; Jenkins, Scanlan, and Peterson 2007; Bundervoet, Verwimp, and Akresh 2009; Akresh and Edmonds 2011). Areas affected by violence are characterized by losses of or poor access to health infrastructures, due to lack of equipment and human resources. Timely interventions in cases of illness are crucial to guarantee full recovery. Limited access to health centers seriously affects antenatal care, professional birth attendance, and postnatal care, thereby endangering the survival of infants. Amid violence, health programs (such as prevention through vaccination and health education) are usually interrupted or implemented discontinuously, increasing the spread of vector-borne diseases. When displacement of large shares of a population occurs, lack of clean water and hygiene leads to a higher risk of diarrhea, one of the major causes of child morbidity and mortality. Finally, infants' health may deteriorate as a consequence of conflict-related shocks experienced in utero (Camacho 2008; Almond and Currie 2011; Akresh, Lucchetti, and Thirumurthy 2012; Mansoor and Rees 2012; Minoiu and Shemyakina 2012).

In line with these intuitive considerations, the literature regularly reports increased infant mortality rates in areas affected by civil war. The detrimental effect is consistent across humanitarian organization reports and the medical literature (Toole, Galson, and Brady 1993; Goma Epidemiology Group 1995; Danish Epidemiology Science Centre 1999; Kiros and Hogan 2001; Médecins sans Frontières 2003; Coghlan et al. 2006). For instance, Coghlan et al. (2006) reported higher infant mortality rates in the eastern side of the Democratic Republic of Congo (DRC), the region of the country most heavily affected by the recent civil war.

Contributions in the demography and economics literature, closer to this

work in scope and methodologies, also consistently find higher child mortality rates in conflict-ridden areas (Guha-Sapir and van Panhuis 2004; Guha-Sapir et al. 2005; Guha-Sapir and D'Aoust 2010). Davis and Kuritsky (2002) showed that severe military conflicts in sub-Saharan Africa raised infant mortality by 12 per thousand. Studying Khmer Rouge Cambodia, de Walque (2005) showed that infant and under-five mortality were very high for children born from 1970 to 1979. In particular, a child born between 1975 and 1979 had roughly a 15 percent risk of dying within the first year of life, with no significant difference in mortality across gender. No gender differences were reported by Singh et al. (2005), who studied child mortality among a displaced population in Sudan and Uganda. Interestingly, they also found no difference in child mortality between refugees and the resident population, hinting that camps for internally displaced people might have an ambiguous effect on mortality. Finally, Verwimp and Van Bavel (2005) found that girls born during the refugee crisis in Rwanda displayed a particularly higher mortality rate as compared with those in the nonrefugee population.

None of the previous studies, however, explicitly controlled for the potential endogeneity of conflict location. In particular, this paper argues that conflict is typically not randomly located and that failing to properly account for this may lead to a bias in the estimated results. The fact that violence has been reported to target wealthier households in neighboring countries like Burundi (Bundervoet 2010), Rwanda (Verpoorten 2009), and Uganda (Blattman and Annan 2010) suggests a bias that may push the estimated response of socioeconomic outcomes to conflict toward zero. Besides the likely nonrandomness of conflict, microlevel data on conflict events (based on news reports) may suffer from measurement error. Conflict events in more remote and less connected locations will typically be underreported in the news and consequently in the data (Restrepo, Spagat, and Vargas 2006; Verpoorten 2012). Controlling for the endogeneity solves these two issues at least partially.

This brings us to the main contributions of the present article. First, it studies the impact of recent violence in DRC on infant mortality rates, explicitly addressing the potential endogeneity of conflict location and timing. It instruments for conflict intensity using a mineral price index. In other words, it exploits the exogenous variation in the potential value of mineral sites generated by changes in world mineral prices to predict the geographic distribution of the conflict. The resulting estimates confirm the concerns expressed above. Ordinary least squares (OLS) results predict (for some specifications of the model) a decrease in mortality rates during the conflict in the districts most heavily affected by the violence, perhaps because the violence targets wealthier households likely to suffer lower infant mortality rates. An instrumental variables

analysis, however, yields the result that conflict increases infant mortality rates, but interestingly the detrimental effect seems to concentrate only among girls. This finding is robust to many different specifications, including a very demanding one that controls for mother fixed effects. The magnitude of the effects is substantial. According to the specification controlling for mother fixed effects (along with a long list of individual and climatic variables), an increase in conflict of 1 standard deviation would translate into a 9 percent increase in the likelihood that a girl will die before she turns one year old.

The second contribution of the current study is to shed light on the gender-specific impact it uncovers. It investigates whether the imbalance is driven by biological or behavioral factors. More specifically, it identifies in the literature potential factors that may foster gender discrimination against girls in households facing difficult times. The analysis finds no evidence for the existence of gender discrimination in the context of DRC conflict. In line with medical evidence (Shettles 1961; Mizuno 2000; Kraemer 2000; Catalano et al. 2006), an alternative explanation relates to the biological vulnerability of boys in utero that would generate gender selection at birth (Valente, forthcoming).[1] The present study assesses whether girls' higher mortality rate may be partially explained by a selection in utero against boys due to conflict exposure, and it finds tentative evidence for this mechanism. Overall, the analysis suggests that gender imbalance in child mortality is largely explained by selection in utero against boys rather than resulting from gender discrimination in times of scarce resources. In other words, the survival of the fittest boys in utero would explain gender imbalances during the first 12 months of life.

In the next section we provide the relevant background information on the armed conflict in the DRC. Section 3 presents the data. Section 4 lays out the empirical strategy, and results are presented in Section 5. Section 6 discusses the relative importance of behavioral and biological factors in explaining the different impact of violence on male and female infants. The last section concludes.

2 Historical Background

The DRC has experienced two of the most violent wars in recent history. The first Congolese war, which started at the end of 1996, is usually interpreted as

[1]The higher vulnerability of boys in utero has been established for other shocks such as famine (Rosebloom et al. 2001; Almond et al. 2008; Hernández-Julián et al. 2014), mother's health deficiencies (Eriksson et al. 2010) or a parental death (Black et al. 2014).

a fight by the coalition of the Congolese rebellion led by Laurent-Desire Kabila with the foreign governments of Rwanda and Uganda not only to overthrow Mobutu but also to eradicate the presence of Rwandan Hutu refugees in eastern DRC, where they had escaped in the aftermath of the 1994 genocide (Vlassenroot and Raeymaekers 2004; Prunier 2009).

The second Congolese war unraveled between 1998 and 2004, with an astonishing estimated death toll of more than 3.8 million people (International Rescue Committee 2011) and an estimated 1.7 million internally displaced people (Internal Displacement Monitoring Center 2011). This magnitude of death and displacement is likely to have impinged upon the health of infants in affected areas. Interestingly, there is extensive anecdotal evidence of the role of minerals in shaping the dynamics of the conflicts, particularly during the second war (Congdon Fors and Olsson 2004; Turner 2007; Gambino 2011; Stearns 2011; Sanchez de la Sierra 2013). This link will constitute the rationale of our instrumental variable strategy.

The provinces most heavily affected by the violence were Orientale and North and South Kivu, the areas in which the concentration of local and foreign armed groups was highest. Conflict was also concentrated in the territory of Pweto (Haut-Katanga district) in the Katanga province as well as in Kinshasa (see Figure 1).

[FIGURE 1 ABOUT HERE]

3 Data Sources and Sample Construction

To assess the impact of the conflict in terms of infant mortality —defined as child mortality at 12 months —we make use of the Demographic and Health Survey (DHS) on DRC carried out in 2007. Since we take advantage of the timing and location variations of conflict events, our main sample excludes children for whom we do not know exactly where their household was living at the time of their birth.[2] We also provide robustness checks extending our sample to an additional wave released in 2014, whose main but critical weakness is to not distinguish between migrant and resident households. We select those

[2]This 2007 DHS dataset provides the number of years a household has been living in the village where the interview took place. Information about previous location is, however, not available, which means that we do not know whether households who migrated remained in the same district. In Section 5 we show that our results are robust to the inclusion of children belonging to nonresident households and to randomly reallocating migrant mothers to districts of origin using a uniform distribution.

children born between 1997 and 2004, encompassing the two Congolese wars. DHS surveys are meant to be nationally representative and collect individual information on women aged 15 to 49 on education, demographic, and health issues as well as some information on the location of the interview, among which are GPS coordinates.[3] Thanks to the inclusion of each woman's maternal history in the dataset, we have recovered detailed information such as when her children were born; whether they are still alive and if not, when they died; and whether they were part of a multiple birth. This enables us to create variables counting the number of brothers and sisters alive at the time of a child's birth.

We also take advantage of the geographical information linked to each DHS cluster to create three climatic variables. Given the emerging evidence on the links between weather shocks and violence (Hsiang, Burke, and Miguel 2013), introducing these variables could potentially reduce the risk of confounding factors. The first two variables are expressed in standard deviations from a long-term average (that is, of the previous 25 years) and are the relative sum of, respectively, monthly rainfall and temperature observations during the first 12 months of life of each child. The data used to construct the measure of precipitation and temperature come from Terrestrial Precipitation: 1900–2008 Gridded Monthly Time Series, Version 2.01, interpolated and documented by Matsuura and Willmott (2009). This dataset is a compilation of updated sources and provides monthly precipitation (and mean temperature) interpolated to a latitude/longitude grid of 0.5 degree by 0.5 degree from an average of 20 weather stations.

We also create a third variable combining rainfall with daily temperature obtained from the Prediction of Worldwide Energy Resource (POWER) database of the US National Aeronautics and Space Administration (NASA): the number of months of potential malaria exposure in the first 12 months of life. To build such an index we apply the approach proposed by Kudamatsu , Persson, and Strömberg (2012). Four conditions have to be simultaneously satisfied for a month to be considered as malaria prone: The malaria index M_{dm} for district d and month m is set to 1 if and only if

1. average monthly rainfall during the past 3 months is at least 60 mm,
2. rainfall in at least 1 of the past 3 months is at least 80 mm,
3. no day in the past 12 months has an average temperature below 5°C, and

[3]Because only surviving women are interviewed, we are likely to underestimate the impact of conflict on child mortality. Nonetheless, there is no reason to believe that this source of sample selection will affect gender imbalances in child mortality.

4. the average temperature in the past 3 months exceeds 19.5°C plus the standard deviation of the monthly temperature in the past 12 months.

For the first stage of our instrumental variables estimates, we investigate the relationship between conflict events and mineral prices. To this end, we construct a panel dataset of conflict events and a price index. We filter the data from the Armed Conflict Location and Event Data Project (ACLED) dataset on the DRC and keep events from January 1, 1997, to December 12, 2004, that are not described as riots (Raleigh et al. 2010). A conflict event is defined as a single altercation wherein force is used by one or more groups for a political end (Raleigh et al. 2010). Thanks to the availability in the dataset of GPS coordinates for each conflict event, we assign each conflict to its respective district and time period, using the shapefiles on DRC from the Global Administrative Areas Database. For each month in the period considered, we create a district-level measure of conflict by summing all events taking place in a given district ($Conflict\,Event$). An alternative source of conflict data from the Uppsala Conflict Data Program (UCDP) is put to use to offer robustness checks.

The location of ore deposits for various minerals, obtained from the mineral occurrences map of DRC, is also assigned to one of the 38 districts.[4] We therefore know the mineral potential of each district of DRC, which we use to compute a price index.

One can observe, in Figures 1 and 2, the geographical repartition by district, respectively, of conflict events from 1997 to 2004 and mineral exploitation sites in DRC. It is rather striking that the eastern part of the country, richer in minerals as confirmed by Figure 2, experienced more conflict events. This correlation, however, does not account for time variations, which we intend to exploit by interacting the mineral potential of each district with the monthly price of the corresponding resource.

[FIGURE 2 ABOUT HERE]

We turn to the United Nations Conference on Trade and Development (UNCTAD) dataset to get the monthly price series of 12 minerals (aluminium, copper, gold, iron, lead, manganese, nickel, oil, phosphate, tin, wolframite, and zinc) and compile information from *The Economics of Tantalum* (Roskill Information Services 2009), Metal Pages, and the US Geological Survey to build our price series for tantalum. The number of potential extraction sites is interacted with the monthly mineral prices to obtain a time-varying measure of relative mineral

[4]The geological service of the African Museum of Tervuren, Belgium, provided the mineral occurrences map of DRC.

value by district ($Price\,Index$). For each time period and district, we compute a price index taking account of the number of mineral deposits and the price for the set of 13 minerals, as follows:

$$Price\,Index_{it} = \sum_{r} \omega_{ri} p_{rt}, \tag{1}$$

where $\omega_{ri} = minerals_{ri} / \sum_j minerals_{rj}$ is a weight measuring the relative importance of mineral r in district i with respect to other districts and p_{rt} is the price of mineral r at period t with a price normalized to 100 for the first period (January 1997). Since we have no information as to the realized or potential extraction of ores of each mineral location, we decided to weigh each monthly ore price by a ratio of the number of mineral deposits in the district over the total number of deposits of this particular mineral in the country. This way, we intend to proxy the extraction potential by ore of each district. We sum this potential over all minerals in a price index in order to reflect the district resource endowment value at each point in time. In other words, we capture how the monthly change in mineral prices alters the relative potential value of the mining sector across districts. This strategy is similar to the one adopted by Bruckner and Ciccone (2010) in their study on conflict in sub-Saharan Africa.[5]

All the variables used in our analysis are described in Table 1. In order to obtain a nationally representative dataset, we resort to sampling weights provided in the DHS. These are needed to render the estimates independent from the sampling design.

Table 1 shows a few noteworthy things. First, infant mortality is relatively high during the period 1997–2004 as 1 child in 10 fails to reach the age of one year. As is commonly observed worldwide, boy mortality is higher than girl mortality (Wilson 1975; Waldron 1998; Garenne 2003). Interestingly, the average ratio of male over female mortality stands at about 1.2, which lies toward the bottom of the range of 1.15—1.30 identified by Garenne (2003) in other countries in normal times. This may already suggest a detrimental impact of warfare on girls' mortality at the aggregated level. Our empirical analysis will put forward compelling evidence that, once the potential endogeneity of conflict location is properly addressed, conflict significantly increases girl mortality.

[TABLE 1 ABOUT HERE]

[5] We also construct a logarithmic version of our price index that produces similar results.

4 Empirical Strategy

The ultimate goal is to estimate the impact of conflict on infant mortality. Since the timing and location of conflict events is likely to be nonrandom and correlated to our dependent variable, we turn to an instrumental variable analysis. We first discuss in detail the first-stage relationship, wherein we predict conflict distribution based on the change in mineral prices. We then use this instrument to assess the impact of conflict on infant mortality.

Conflict

In the context of conflict in the DRC, we expect resources to shape the distribution of violence. We therefore use the previously described price index to predict the intensity of conflict by district and over time. Formally, the first-stage specification is as follows:

$$Conflict_{dt} = \alpha_d + \beta_t(+\delta_{dt}) + \gamma Price\,Index_{dt} + \varepsilon_{dt}, \tag{2}$$

where $Conflict_{dt}$ is one of the measures of conflict. We control for district fixed effects, α_d, time fixed effects, β_t, and, in some specifications, district-specific linear time trends, δ_{dt}. We run several specifications of the model using *Conflict Events* as dependent variable to check the robustness of the first-stage relationship. Next, given the interest of this study, we run the first stage to predict, for each month in the sample, the conflict distribution over the preceding 12 months, denoted by *Conflict Events* 12. We also adopt an alternative measure of conflict, which records for each month the number of months featuring violence in the districts over the preceding 12 months, denoted by *Conflict Exposure* 12. In those cases, we change the price index used accordingly (*Price Index* 12).

Infant Mortality

The second stage of the analysis focuses on infant mortality, defined as the mortality of children by 12 months of age. In order to exploit the richness of the dataset, we first run cross-sectional regressions with several types of fixed effects, including a within-household comparison through the use of mother fixed effects. As an alternative specification, we next run panel regressions to estimate mortality rates at 12 months by district.

Cross-section Regressions

In this context, the unit of observation is a child, i, born at month m, in district d. We check 12 months after her birth whether she is still alive and assign value 1 to our binary variable $Mortality_{imd}$ if child$_i$ died during the first 12 months of her life. We denote by $Conflict\,Events\,12_{dm}$ the sum of conflict events that took place in district d during the first 12 months of life of child i born in month m, that is $Conflict\,Events\,12_{dm} = \sum_{j=0}^{11} Conflict\,Events_{dm+j}$.
Formally, we estimate the following model:

$$Mortality_{idm} = \alpha_d + \beta_m + \delta^{'} X_i + \lambda^{'} X_{hh} + \gamma Conflict\,Events\,12_{dm} + \varepsilon_{idm}, \quad (3)$$

where X_i is a vector of control variables including whether the child was part of a multiple birth, whether she was the first child, her number of siblings alive, a malaria index (summing the number of months of exposure to malaria), and rainfall and temperature anomalies (with respect to a long-term average of 25 years). A further set of variables, X_{hh}, controls for household characteristics including the education level of the mother and a measure of the wealth of the household.

To deal with the potential endogeneity of conflict distribution, the conflict intensity measure is instrumented by the mineral price index described above. Formally, the following system of two equations is estimated by limited information maximum likelihood:

$$Mortality_{imd} = \alpha_d + \beta_m + \delta^{'} X_i + \lambda^{'} X_{hh} + \gamma Conflict\,Events\,12_{dm} + \varepsilon_{idm} \quad (4)$$

$$Conflict\,Events\,12_{dm} = \delta_d + \rho_m + \eta^{'} X_i + \theta^{'} X_{hh} + \nu Price\,Index\,12_{dm} + \upsilon_{idm} \quad (5)$$

The idea behind this empirical strategy is to exploit the timing and location variations of conflict events and to compare children born in the same month in districts affected differently by conflict. Standard errors are clustered at the district level, and sampling weights are used to render the estimates independent of the sampling design. Given the small number of clusters ($n = 38$) which might produce understimated intra-group correlation, we turn to 999 replications of wild bootstrap (percentile-t method), known to resist to heteroskedasticity, to compute confidence intervals. As mentioned before, the regressions are run on the sample of children known to have been born in their mother's interview district. Enlarging the sample to include those children who migrated does not qualitatively change the results.

The most demanding and most convincing specification of this empirical exercise is to include in the model mother fixed effects, enabling us to compare along the dimension of exposure to conflict children born to the same mother.

Controlling for mother fixed effects allows us to take account of both environmental differences between children and their mother's genetic features. In order to ease the estimation process, we partial out each variable with the series of all the fixed effects. By the Frisch-Waugh-Lovell theorem, we know this method to keep the sign and magnitude of the coefficients unchanged while inference might be marginally influenced. This is an additional reason to again turn to wild bootstrap to produce confidence intervals.[6]

Beyond the power of the instruments, this empirical strategy is based on a key identifying assumption. If mineral prices were to influence infant mortality through another channel than the occurrence of violence, this would violate our exclusion restriction. In particular, one could claim that wealth effects coming from mineral price variations could invalidate our identification strategy. A first indication that it should not be the case is that our point estimates are roughly unchanged when controlling for household characteristics such as the education of the mother or household wealth. Controlling for wealth effects, the exclusion restriction is likely to hold. To test further the plausibility of our exclusion restriction, we proceed to a placebo test. Regressions of the same price index on infant mortality over the period 1980–1996 showed that the exclusion does not seem to be at risk.[7] In particular, we fix the length of the sample to 96 months, as in the main analysis. Next, we run regressions starting on the first month of the enlarged sample (January 1980). Moving each time by one month our sliding window of 96 months from the start of the sample until the end, we test the reduced-form relationship in 95 regressions for the preconflict period. When we include no district-specific linear trends, we obtain only 9 out of 95 times a (weakly) significant reduced-form relationship. Including linear district-specific trends yields no significant coefficient out of the 95 regressions. The results of this placebo exercise are summarized in Table 2. Such a result is difficult to conciliate with price-induced wealth effects.

[TABLE 2 ABOUT HERE]

Panel Regressions

As an alternative specification, we aggregate the data at the district level (at the cost of not controlling for changes in the composition of the district populations). The unit of observation is then the district, d, for which we compute

[6]Traditional robust two-stage least squares standard errors produced qualitatively similar results.

[7]Infant mortality for this period is reconstructed retrospectively from the DHS.

a mortality rate on all the children born there 12 months before, $Mortality_{dm}$.

We run the following fixed-effect panel regression (with or without district-specific linear trends):

$$Mortality_{dm} = \alpha_d + \beta_m + \gamma Conflict\,Events\,12_{dm} + \varepsilon_{dm}. \tag{6}$$

As for the cross-section analysis, we correct for endogeneity with two-stage least squares, instrumenting conflict by mineral prices, and estimate the following system of equations:

$$Mortality_{dm} = \alpha_d + \beta_m + \gamma Conflict\,Events\,12_{dm} + \varepsilon_{dm} \tag{7}$$

$$Conflict\,Events\,12_{dm} = \delta_d + \rho_m + \eta Price\,Index\,12_{dm} + \epsilon_{dm}. \tag{8}$$

5 Results

Conflict

Table 3 exhibits that at the district level, the relationship between mineral prices and conflict events is highly significant and negative (first column and row of Panel A). Adding linear district-specific time trends confirms the significance of the price index coefficient (first column and row of Panel B). We run a set of further robustness checks on the first stage. In the second row of column 1, we exclude tantalum from the price index, given the alleged relevance of this mineral in the conflict dynamics as stressed in the international press. Conversely, in the third row we compute the price index only on tantalum. Interestingly, removing one mineral at a time from the analysis does not alter the results. The next three rows replicate the first three but predict conflict in a 12-month period based on the sum of the price variations over a 12-month period.

In column 2, we restrict the analysis to the 19 districts (half of DRC districts) hosting most of the violence in the period considered. Here also, removing one district at a time does not qualitatively change the findings. In column 3 we restrict the analysis to the 19 districts richest in minerals (based on the price index).

[TABLE 3 ABOUT HERE]

In column 4–6 we focus on the role of tantalum in the relationship between mineral prices and conflict. In column 4 the analysis is conducted only on districts with tantalum deposits. Column 5 restricts the sample to the 5 districts most influenced by the tantalum price changes, and the last column looks at

the districts excluded from column 5. All specifications confirm the negative relationship.

Finally, we test the first stage using a nonparametric smoother (a locally weighted smoother, specifically the lowess estimator) on the demeaned and detrended versions of the conflict and mineral price measures. The result of this exercise, reported in Figure 3, confirms that the linear specification constitutes a good approximation of a potentially more complex relationship.[8] The negative relationship is again confirmed using a nonparametric approach.

[FIGURE 3 ABOUT HERE]

Table 4 puts forward our measures of conflict, *Conflict Events* 12 and *Conflict Exposure* 12, the coefficient of which is again strongly significant and negative. Significance is actually increased with linear district-specific trends.[9]

[TABLE 4 ABOUT HERE]

Infant Mortality

Table 5 presents cross-section regressions over the sample of children born between 1997 and 2004, for all children (columns 1–2), only boys (columns 3–4) or only girls (columns 5–6). We find strong evidence for the nonrandom distribution of violence. Table 5 suggests a bias to zero of 'naive' OLS regressions. These biases affecting the OLS estimates suggest that some unobserved factors simultaneously explain the number of conflict events by district and child mortality. A potential explanation may be that conflicts are more likely to target wealthier (and therefore healthier or more able to cope with adverse shocks) households in a looting-driven warfare. These biases may also arise from measurement errors. The results also identify a gender-specific impact of conflict episodes on child mortality. Girls are more adversely affected by conflict than boys. According to column 6, a change by 1 standard deviation in the number of conflict events (that is, by about 14 conflict events) increases the probability of a girl's dying within the first year of life by about 7 percent. The magnitude of this effect is far from trivial, inasmuch as it constitutes a doubling of girls mortality (at mean value). Adding the full set of controls in Panel B of Table 5 leaves the coefficients of interest virtually unchanged (slight increase of the coefficient and the power of our instrument). That is very reassuring with respect

[8] Reducing the bandwidth of this estimator from 0.80 to 0.20, and hence producing less smoothing by focusing on closer points in the local regressions, leaves the general trend unchanged.

[9] These results hold if we use quadratic instead of linear trends.

to the exclusion restriction. Conditional on household wealth, the exclusion restriction is more likely to hold.[10] Given the magnitude and significance of the F-statistics on the instrument, it is very unlikely that the analysis should be invalidated by a problem of weak instrument.

[TABLE 5 ABOUT HERE]

Being part of a multiple birth is robustly significant and increases mortality, although the result hinges on only 2 percent of the sample. Being a first child is likely to be detrimental as well, although the impact is less robust because it loses significance once the sample is split by gender, following the increase of the standard errors. In line with the literature, we find strong and robust harmful effects for children whose mother does not record any years of formal education (see, for instance, Behrman and Wolfe 1987; Strauss and Thomas 1995). As expected, wealth decreases infant mortality. These results are quite robust to alternative specifications. Household size is also negatively associated with infant mortality. In Table 6, we assess the robustness of the results to the addition of a linear trend (column 2), the inclusion of nonresident households in the sample (column 3), and the addition of the additional wave of the DHS respectively with and without linear trend (columns 4–5). It is to be noted that adding controls increases the precision and reduces the noise in our estimates. The addition of a linear trend worsens the efficiency of the point estimates while, as expected, the precision and the size of the conflict coefficients decrease with the extended sample. As expected, including observations from the 2013 DHS, brings addtional noise to our estimates lowering the magnitude and the significance of the coefficient of interest.[11]

[TABLE 6 ABOUT HERE]

Despite the addition of mother and household characteristics in Panel B of Tables 5 and 6, we cannot be certain unobserved child characteristics are not driving the relationship between violence and infant mortality. To reduce that concern, we introduce mother fixed effects. Intuitively, we compare the

[10]Figure A.1 in appendix further supports the exclusion restriction. It graphically shows the coefficients obtained by regressing girl mortality on the price index over a moving window of 96 months. It is striking to see how the coefficients are consistently around zero in the preconflict period. The negative response of girls' mortality to mineral price shocks is particularly strong during the period of investigation. As shown on the right panel, the relationship is even stronger in highly conflictive districts.

[11]Turning to an alternative source of conflict data, UCDP, qualitatively confirms our results. See Table A.1. in the Appendix

mortality of siblings differently exposed to conflict, by taking account of their shared environment and common genetics. The gender imbalance in infant mortality is further confirmed in Table 7. The magnitude of the effect remains sensibly the same while, once we control for mother fixed effects, other controls seem to be much less influential. According to the results reported in column 6, an increase in conflict of 1 standard deviation would translate into a 9 percent increase in the likelihood of a girl's dying before she turns one year old. Said differently, an increase of 1 standard deviation in conflict would be responsible for the death of 397 additional girls out of the sample of 4409 girls. As for the previous specifications, the mother fixed effects results do not depend on the inclusion of climate and individual characteristics (Panel B of Table 7). Table 8 further indicates that the results are robust to the inclusion of a district-specific time trend, the inclusion of nonresident households in the sample, and the inclusion of observations from the 2013 wave of the DHS (respectuvely with and without linear trends).

[TABLE 7 ABOUT HERE]
[TABLE 8 ABOUT HERE]

The inclusion of these observations exacerbates the problems posed by migrations which could bias our results. One could indeed suspect that households would flee from conflict prone districts to safer locations. Descriptive statistics show however that while the 5 districts the most prone to conflict account for 26.4% of the total number of households, they represent precisely 1/3 of all the migrant households. In addition to that, the proportion of migrant households in the whole sample is 18.4% while it is 22.1% in these particular districts. It therefore seems that even though these districts accomodate proportionaly more migrants than the others, they seem to move inside these districts which is taken into account by our extended sample regressions. Still, we investigate further this issue by turning to simulations and randomly allocating migrant mothers to any of the 38 districts. Gender imbalances in child mortality are confirmed when randomly reallocating migrant mothers to districts of origin using a uniform distribution. In 71.8 percent of the 1000 simulated samples, the impact of conflict on girl mortality is positive and significant at, at least, 90 percent level of confidence while it is significant at the 95% level of significance in 53.1%. [12]

[12]Given the lack of information about the district a migrant household resided when migrating, we had little choice but to perform a simulation exercise. We run 1000 two-stage least-squares panel regressions (on boy and girl mortality) where each migrant mother is randomly allocated to one of the 38 districts, following a uniform distribution (with equal probability to migrate in any district).

As a final robustness test we aggregate the data at the district level and run panel regressions on mortality rates by gender. The results, presented in Table 9, are qualitatively identical to those obtained from cross-section regressions using individual observations. According to the estimate reported in column 6 of Panel A, an increase by 1 standard deviation (12.3) in conflict events would increase the likelihood of a girl's dying within the first 12 months of life by about 7.1 percentage points. That would correspond to an increase of about 94 percent, at mean value. Such an increase is actually almost identical to the one obtained with the cross-sectional estimates but lower than our preferred specification with mother fixed effects. As in the context of cross-section analysis, these results remain unaffected by the inclusion of aggregated climatic variables (Panel B of Table 9).

[TABLE 9 ABOUT HERE]

The results obtained in this section consistently suggest that considering conflict events as nonrandom events and consequently exploiting mineral price variations as exogenous shocks on the likelihood of conflicts makes a significant difference when estimating the impact of conflict on infant mortality. A robust pattern emerges: when we control for the endogeneity of conflict intensity, we find that girls are substantially more affected by violence than boys. The next section attempts to identify the mechanisms behind this gender-specific effect.

6 Behavioral versus Biological Factors

Two broad classes of factors may drive these findings. First, households may discriminate against girls when resources within households become more limited during times of warfare. In other words, gender imbalances in infant mortality would be the result of behavioral factors in a situation in which sons are favored by parents.[13] An alternative explanation relates to the biological vulnerability of boys in utero that would generate a strong selection effect at birth. As explained by Valente (forthcoming), gender-based mortality selection in utero may be explained either by the fact that a male fetus is frailer than its female counterpart (Shettles 1961; Kraemer 2000; Mizuno 2000; Catalano et al. 2006) or by the ability of females in poor conditions to favor female offspring due to the

[13]Another possibility would be that girls have been systematically targeted by armed violence. Even though gender-based (sexual) violence has been a dramatic phenomenon in eastern DRC (Peterman, Palermo, and Bredenkamp 2011), since we are dealing with infants, this is not likely to be the major mechanism explaining gender imbalances.

lesser variance in reproductive success of girls compared with boys, the so-called Trivers and Willard (1973) hypothesis. Distinguishing behavioral factors from biological ones is far from obvious from an empirical point of view (Garenne 2003; Mu and Zhang 2011). In this section, we propose various strategies to test the plausibility of the two mechanisms.

We conjecture that the behavioral hypothesis would be consistent with several exacerbating or mitigating factors. The results of tests for behavioral factors are reported in Table 10, where only the coefficients of interest are presented for different specifications. First, the literature on gender discrimination suggests that when the decisionmaking power is in the hands of women, such discrimination against girls should be at least partly corrected (Thomas 1990, 1994; Duflo 2003, 2012). Interacting the conflict measure with a dummy capturing female-headed households, we do not find any evidence for such a mitigating factor (Panel A of Table 10). In contrast, we could also expect widows to particularly value the survival of a male offspring in a context in which inheritance rules are generally discriminatory against women. If this is true, we should observe more discrimination against girls of widows during violence. Interacting conflict with a dummy for widows, however, no differentiated impact is found (Panel B).

[TABLE 10 ABOUT HERE]

Consistent with the literature on sibling rivalry (Morduch 2000; Akresh and Edmonds 2011), we expect the composition of the household, and in particular the number of brothers and sisters to have an additional impact on girls' mortality. Controlling for household size, when a girl has several brothers, we expect gender discrimination to be even stronger. The reverse is expected when a girl has several sisters, because the burden of gender discrimination would be shared among sisters. The results of these exercises, reported in Panels C and D, suggest once more that the behavioral hypothesis has very little explanatory power.[14]

An alternative strategy to assess the role of behavioral factors consists in introducing an interaction term between violence and a proxy for son preference at the household level. We define boy preference following Jayachandran and Kuziemko (2011) in subtracting the ideal number of sons reported by each mother to the number of boys alive in her household. If our gender imbalance is driven by discrimination, we should find a stronger discrimination among households whose boy preference is stronger. The results of this test, in Panel

[14] Controlling for the number of siblings similar to Akresh and Edmonds (2011) or using a dummy for having at least one brother or one sister does not change the results.

E, show no additional effect due to stronger boy preference. We also test the robustness of these results to dividing the Jayachandran and Kuziemko (2011) proxy for son preference by the same indicator for daughter preference, to disentangle the preference for boys from the preference for large family. No evidence of stronger probability of girls' dying within the first year of life is found in households with a stronger boy preference. That remains true when we adopt a different measure for boys preference, namely the ratio between the number of boys alive and the ideal number of sons reported by mothers.[15] Since we do not find any evidence for the existence of heterogeneous impacts along the dimensions that should reveal the presence of gender discrimination, we conclude that the behavioral hypothesis is unlikely to constitute a major explanation for the gender imbalances in infant mortality due to the civil war in DRC. Of course, the absence of evidence is only suggestive of an evidence-of-absence effect. But at least, contrary to initial expectation, the behavioral factors seem not to be driving the gender imbalances in mortality in times of conflict in DRC.

The main alternative explanation for the gender-specific effect hinges on a biological hypothesis. As reported above, medical evidence shows that male fetuses are more vulnerable than their female counterparts. In particular, recent studies have provided strong evidence that the sex ratio at birth decreases following a worsening of the pregnancy environment, as a consequence of a civil war (Valente, forthcoming), terrorist attacks (Catalano et al. 2006), or prolonged economic crisis (Fernandez et al. 2011). If this has been the case in DRC, the higher impact of violence on girls' mortality rate may be a direct consequence of the selection induced by higher mortality of boys in utero. We therefore test whether conflict intensity affected the sex ratio at birth in the panel framework. To reduce the number of undefined values for the ratio when either no boy or no girl is born in a particular month, we define the sex ratio for each region and district as the number of boys born over the total number of births (replacing missing values by 0.5 when no birth is recorded for a particular month). The average ratio in our sample stands at 0.511, which is slightly higher than half the value of 1.03 of the sex ratio (boys over girls) at birth given by Anderson and Ray (2012) for the whole sub-Saharan Africa (compared with 1.06 in developed countries). We find some tentative evidence, reported in Table 11, that the sex ratio at birth significantly decreases following violence experienced during pregnancy. According to the results reported in column 2 of Panel A, an increase by 1 standard deviation in the number of violent events experienced in utero reduces the sex ratio by about 14 percent, at the mean value of the sex ratio at birth. Using the alternative measure of conflict (months of exposure) provides

[15]Results not reported in the paper; available upon request.

very similar results (column 4).

[TABLE 11 ABOUT HERE]

To further investigate the strength of the selection effect in utero, we assess the impact of violence experienced in utero and during the first 12 months of life on the sex ratio among one-year-old children (Panels B–D of Table 11). We first look at the impact of violence experienced while in utero on the sex ratio among one-year-old children. Results turn (barely) insignificant but are still negative (Panel B). We then assess the separate impact of violence in utero versus violence during the first 12 months of life on sex ratio in Panel C. Finally, in Panel D, we test the cumulative impact of violence from conception until the first birthday on sex ratio. The results jointly confirm the importance of violence experienced in utero in driving the sex ratio. The higher mortality rate among girls caused by violence during the first 12 months of life, as documented in this study, is only marginally compensating for the strong selection effect against boys in utero.

In the absence of evidence for behavioral factors, this analysis suggests that gender imbalances in infant mortality are at least partially driven by the selection induced by higher vulnerability of boys in utero.

7 Conclusions

In this paper we analyzed the impact on infant mortality of the armed conflict afflicting the DRC from 1997 to 2004. This study differs from existing microlevel studies in a major way. Relying on a credible instrumental variable approach, we control for the nonrandom timing and location of conflict violence. This is particularly relevant when we exploit within-district variations inasmuch as we show that our instrumental variables results significantly differ from our ordinary least squares findings, raising some concerns about potential bias in the existing studies relying on geographical variation in exposure to conflicts.

We find that experiencing violence substantially increases mortality rates among infants, but only for girls. This pattern is robust to many different specifications, controlling for district and month fixed effects, rainfall anomalies, and mother fixed effects. Why does violence affect especially girls' mortality? Two broad classes of factors could explain the gender imbalance we uncover: behavioral and biological. According to the former, girls may be more discriminated against by households faced with difficult circumstances in times of conflict, to safeguard their male offspring. The latter, instead, would ascribe to purely biological factors the gender-specific resilience to conflict. For instance, boys and girls may feature different resistance levels to negative shocks.

We adopt several strategies to assess the different roles of behavioral versus biological factors in explaining our result. We find no support for gender discrimination to be driving the higher mortality rates among girls. Instead, we find suggestive evidence that gender imbalances in infant mortality in times of warfare are mainly driven by biological factors. More specifically, we show that violence is more fatal for male fetuses than for their female counterparts. In turn, the higher vulnerability of boys in utero induces a selection in the sample of children born in conflict-affected regions. Overall, our results suggest that more attention should be paid to understanding possible selection in utero in studies assessing the impact of shocks in early life.

Our analysis also delivers a critical policy recommendation. Gender-specific warfare damages have sometimes led scholars and policymakers to call for gender-based targeted interventions. Although they may be grounded on good motives and may help in reducing gender discrimination in general, our study warns that these policies may miss their targets if they fail to account for the possible selection in utero. In other words, despite providing some evidence of gender imbalances in infant mortality, our paper suggests that any policy should be drawn on a sound understanding of the sources of such gender bias. As biological factors in utero are found to be a more prominent explanation than the standard behavioral factors, our paper resets the priority to policies aiming at enhancing the resilience of (pregnant) women to violent experiences. Policies ensuring high coverage of multiple micronutrient supplementation and other nutrition-sensitive programs directly addressing pregnant women (like cash transfers), family planning to delay the age of first pregnancy, or educational interventions designed to increase spacing between births may therefore all prove comparatively more effective in reducing infant mortality in times of violence than policies targeting gender discrimination during the first year of life (Black et al. 2013; Bhutta et al. 2013; Ruel and Alderman 2013; WHO 2014).

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Table 1: Descriptive statistics (1997–2004)

Variable	N	Mean	Std. dev.	Min.	Max.
Panel A: Cross-section dataset					
Infant mortality	10,397	0.107	0.310	0	1
Boy mortality	5,242	0.117	0.321	0	1
Girl mortality	5,155	0.098	0.297	0	1
Number of conflict events	10,397	7.896	14.467	0	123
Exposure to conflict (# months)	10,397	2.276	2.820	0	12
Price index 12 m	10,397	492.04	624.06	0	3,002.6
Twin	10,397	0.017	0.131	0	1
1st child	10,397	0.195	0.396	0	1
# brothers alive	10,397	1.209	1.457	0	8
# sisters alive	10,397	1.175	1.430	0	8
Exposure to malaria	10,397	7.4	2.991	0	11
Rain 25y (dev)	10,397	-0.239	0.990	-3.477	5.149
Temp 25y (dev)	10,397	0.737	1.083	-1.546	5.397
Mother —no educ.	10,397	0.251	0.434	0	1
Mother —primary	10,397	0.424	0.494	0	1
Mother —secondary	10,397	0.315	0.464	0	1
Wealth index	10,397	-1,655.5	97,422.5	-107,521	341,565
Female-headed HH	10,397	0.139	0.346	0	1
Household size	10,397	7.336	3.063	1	28
Panel B: Panel dataset (district-month)					
Infant mortality rate (12 m)	2,876	0.106	0.221	0	1
Boy mortality rate (12 m)	2,876	0.090	0.234	0	1
Girl mortality rate (12 m)	2,876	0.076	0.218	0	1
Number of conflict events	2,876	5.639	12.327	0	123
Exposure to conflict	2,876	1.769	2.538	0	12
Price index 12 m	2,876	417.351	585.228	0	3,002.567
Months of malaria exposure (avg)	2,876	7.699	2.666	0	11
Rain 25 y (avg dev)	2,876	-0.213	0.944	-3.239	5.149
Temp 25 y (avg dev)	2,876	0.826	1.096	-1.523	5.259
Sex ratio at birth	2,598	0.511	0.350	0	1
Number of conflict in utero	2,598	4.412	10.079	0	103
Exposure to conflict in utero	2,598	1.353	1.951	0	9
Price index 9m in utero	2,598	296.009	412.647	0	1,924.32

Notes: Correction for sampling weights. HH=household.

Table 2: Placebo test of exclusion restriction

p-value less than:	0.1	0.05	0.01	# regressions
No trends	9	0	0	95
Linear trends	0	0	0	95

Notes: The table reports the number of times we found a significant reduced-form relationship between our price index and infant mortality for the prewar period 1980–1996 using a sliding window of 96 months for each regression.

Table 3: Panel regressions on the impact of mineral prices on conflict

Dependent variable:	Monthly conflict events					
	(1)	(2)	(3)	(4)	(5)	(6)
	All	19 highest C	19 highest P	TA districts	5 districts	Drop (5) districts
Panel A: No trend						
Price index	-.00922***	-.0107***	-.0102***	-.0125***	-.0226***	-.00498*
	(.00224)	(.00271)	(.00297)	(.00234)	(.0033)	(.00262)
Price index - No TA	-.00892***	-.0106***	-.00902***	-.0225*	-.0467*	-.00582**
	(.00281)	(.00341)	(.00246)	(.0118)	(.0205)	(.00245)
TA price index	-.0106**	-.012**	-.0129**	-.0114**	-.0145	.151
	(.0044)	(.00509)	(.00493)	(.00471)	(.00931)	(.119)
Price index 12	-.012***	-.0154***	-.0151***	-.0218***	-.0415**	-.00406
	(.00391)	(.0048)	(.00496)	(.00393)	(.0108)	(.00251)
Price index 12 - No TA	-.00618**	-.00874**	-.00896**	-.00755	-.039*	-.00441*
	(.00256)	(.00343)	(.00312)	(.0107)	(.0166)	(.00251)
TA price index 12	-.0298***	-.0303***	-.0322***	-.0309***	-.0445**	.144
	(.00554)	(.00619)	(.00596)	(.00591)	(.0124)	(.118)
Panel B: Linear trend						
Price index	-.0103***	-.0116***	-.0105***	-.0125***	-.0171**	-.0073**
	(.00198)	(.00233)	(.00283)	(.0022)	(.00543)	(.00331)
Price index - No TA	-.0133***	-.0154***	-.011***	-.0328**	-.0362	-.00866***
	(.00373)	(.0043)	(.00333)	(.0143)	(.0332)	(.0026)
TA price index	-.00906**	-.0103**	-.0113**	-.00983**	-.0115	.151
	(.00406)	(.00476)	(.0046)	(.00437)	(.00863)	(.12)
Price index 12	-.0167***	-.0189***	-.0177***	-.0251***	-.0292	-.00638**
	(.00358)	(.00315)	(.00353)	(.0036)	(.0144)	(.00298)
Price index 12 - No TA	-.0124***	-.0156***	-.0116***	-.0286***	-.025	-.00719**
	(.00395)	(.00424)	(.00394)	(.00939)	(.0251)	(.00276)
TA price index 12	-.0245***	-.0244***	-.0266***	-.0256***	-.0362***	.152
	(.00298)	(.00371)	(.00344)	(.00339)	(.00759)	(.117)
N	3648	1824	1824	2112	480	3168
District FE	✓	✓	✓	✓	✓	✓
Month-year FE	✓	✓	✓	✓	✓	✓

Notes: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$; FE = fixed effects; TA = tantalum.

Table 4: First-step specifications

Dependent variable:	Conflict events 12		Conflict exposure 12	
Trend:	None	Linear	None	Linear
	(1)	(2)	(3)	(4)
Price index 12	-.0120***	-.0167***	-.0014*	-.0022***
	(.0039)	(.0036)	(.0007)	(.0006)
N	3,648	3,648	3,648	3,648
District FE	✓	✓	✓	✓
Month-year FE	✓	✓	✓	✓

Notes: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$; FE = fixed effects.

Table 5: Regressions of intensity of conflict on child mortality

Dependent variable:	Mortality rate at 12 months					
	All		Boys		Girls	
	OLS	2SLS	OLS	2SLS	OLS	2SLS
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: No controls						
Conflict events 12	-0.0008	0.0003	-0.0019***	-0.0035	0.0003	0.0054*
	(-0.002 - 0.000)	(-0.0123 - 0.0128)	(-0.003 - -0.0007)	(-0.0116 - 0.0046)	(-0.0027 - 0.0032)	(-9.35e-05 - 0.011)
Kleibergen-Paap rk Wald F		17.32		12.72		22.26
Panel B: Full set of controls						
Conflict events 12	-0.0008	0.0016	-0.0018***	-0.0017	0.0002	0.0066**
	(-0.002 - 0.0003)	(-0.0045 - 0.0076)	(-0.0029 - -0.0006)	(-0.0107 - 0.0072)	(-0.0023 - 0.0027)	(0.0001 - 0.0131)
Twin	0.163**	0.163**	0.106*	0.107*	0.238*	0.239
	(0.0389 - 0.287)	(0.0391 - 0.288)	(-0.0142 - 0.226)	(-0.00794 - 0.223)	(-0.0337 - 0.509)	(-0.0492 - 0.527)
1st child	0.0120	0.0131	0.0216	0.0223	0.0018	0.005
	(-0.0112 - 0.0352)	(-0.0129 - 0.0391)	(-0.0192 - 0.0624)	(-0.0181 - 0.0627)	(-0.0400 - 0.0436)	(-0.0295 - 0.0394)
# brothers	-0.0035	-0.0029	0.0022	0.0024	-0.0084	-0.0068
	(-0.0115 - 0.0046)	(-0.0113 - 0.0055)	(-0.0091 - 0.0136)	(-0.0100 - 0.0148)	(-0.0193 - 0.0024)	(-0.0165 - 0.0029)
# sisters	0.0096	0.0094	0.006	0.0063	0.0120	0.0116
	(-0.0104 - 0.0297)	(-0.0125 - 0.0313)	(-0.0558 - 0.0679)	(-0.0357 - 0.0482)	(-0.0057 - 0.0297)	(-0.0066 - 0.0297)
Mother no educ.	0.0446	0.0444	0.0166	0.0166	0.0759**	0.0748***
	(-0.0224 - 0.112)	(-0.0193 - 0.108)	(-0.100 - 0.133)	(-0.0949 - 0.128)	(0.0167 - 0.135)	(0.0195 - 0.130)
Mother primary	0.0082	0.0076	-0.0117	-0.0125	0.0282	0.0257
	(-0.0347 - 0.0512)	(-0.0271 - 0.0423)	(-0.0923 - 0.0689)	(-0.0996 - 0.0746)	(-0.0096 - 0.066)	(-0.0107 - 0.0622)
Mother secondary	-0.0203	-0.0209	-0.0496	-0.0505	0.008	0.0057
	(-0.0597 - 0.0192)	(-0.0573 - 0.0156)	(-0.140 - 0.0404)	(-0.149 - 0.0478)	(-0.00991 - 0.0259)	(-0.0121 - 0.0234)
Wealth index	-6.39e-08	-6.31e-08	-6.92e-08	-6.28e-08	-4.40e-08	-4.48e-08
	(-2.19e-07 - 9.14e-08)	(-2.23e-07 - 9.71e-08)	(-3.05e-07 - 1.67e-07)	(-2.50e-07 - 1.24e-07)	(-3.31e-07 - 2.43e-07)	(-4.03e-07 - 3.13e-07)
Female headed HH	-0.0156	-0.0157	-0.0247**	-0.0257**	-0.0086	-0.008
	(-0.0456 - 0.0143)	(-0.0467 - 0.0153)	(-0.0486 - -0.000895)	(-0.0481 - -0.00339)	(-0.0631 - 0.0460)	(-0.0508 - 0.0349)
HH size	-0.007**	-0.007**	-0.0071**	-0.0071**	-0.0068***	-0.0067**
	(-0.0129 - -0.0011)	(-0.0124 - -0.0015)	(-0.0138 - -0.0003)	(-0.0138 - -0.00047)	(-0.0119 - -0.0018)	(-0.0122 - -0.0012)
Malaria exposure	0.0017	0.0013	-0.0004	-0.0002	0.0035	0.0023
	(-0.0085 - 0.0119)	(-0.0083 - 0.0108)	(-0.006 - 0.0052)	(-0.0088 - 0.0083)	(-0.0046 - 0.0116)	(-0.0077 - 0.0124)
Rain 25y (dev)	0.0019	0.0004	0.0018	0.001	0.0028	-0.0006
	(-0.0131 - 0.0168)	(-0.0201 - 0.0208)	(-0.0242 - 0.0278)	(-0.0185 - 0.0205)	(-0.0513 - 0.0570)	(-0.0447 - 0.0436)
Temp 25y (dev)	0.0084	0.0083	0.0189	0.0195	0.0012	-0.0002
	(-0.0094 - 0.0263)	(-0.0083 - 0.0249)	(-0.0076 - 0.0453)	(-0.0047 - 0.0436)	(-0.0279 - 0.0303)	(-0.0157 - 0.0153)
Kleibergen-Paap rk Wald F		18.69		13.97		23.33
District FE	✓	✓	✓	✓	✓	✓
Month-year FE	✓	✓	✓	✓	✓	✓
N	8,869	8,869	4,460	4,460	4,409	4,409

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1; correction for sampling weights; sample of residents only; Standard errors clustered at the district level and confidence intervals produced by wild bootstrap (percentile-t method).

Table 6: 2SLS cross-sectional regressions on girl mortality - alternative specifications

Dependent variable:	Girls' mortality at 12 months				
	2SLS	2SLS	2SLS	2SLS	2SLS
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Conflict events 12	0.0054*	0.0053	0.0041	0.0026	0.0024
	(-0.000 - 0.011)	(-0.001 - 0.012)	(-0.001 - 0.009)	(-0.001 - 0.006)	(-0.001 - 0.006)
Kleibergen-Paap rk Wald F	22.26	22.44	15.28	18.37	21.85
Panel B: Full set of controls					
Conflict 12	0.0066**	0.0061*	0.0053*	0.0033*	0.0030*
	(0.0005 - 0.0127)	(-0.0003 - 0.0125)	(-0.0002 - 0.0109)	(-0.0002 - 0.0067)	(-0.0005 - 0.0065)
Twin	0.239	0.234	0.190*	0.155***	0.153***
	(-0.0769 - 0.555)	(-0.0783 - 0.546)	(-0.0338 - 0.414)	(0.155 - 0.155)	(0.153 - 0.153)
1st child	0.00496	0.0044	-0.0002	-0.0146*	-0.0146*
	(-0.0368 - 0.0467)	(-0.0326 - 0.0414)	(-0.0132 - 0.0128)	(-0.0300 - 0.0008)	(-0.0299 - 0.0007)
# brothers	-0.0068	-0.0071	-0.0039	-0.0074***	-0.007**
	(-0.0167 - 0.0031)	(-0.0168 - 0.0027)	(-0.0145 - 0.0068)	(-0.0130 - -0.0018)	(-0.0128 - -0.0012)
# sisters	0.0116	0.0122	0.00849	0.00359	0.00348
	(-0.0064 - 0.0295)	(-0.0074 - 0.0317)	(-0.0071 - 0.0241)	(-0.0067 - 0.0139)	(-0.0074 - 0.0143)
Mother no educ.	0.0748***	0.0776***	0.0817***	0.0511*	0.0498*
	(0.0274 - 0.122)	(0.0284 - 0.127)	(0.0234 - 0.140)	(-0.003 - 0.105)	(-0.0067 - 0.106)
Mother primary	0.0257	0.0291	0.0367**	0.0331	0.0318
	(-0.00793 - 0.0594)	(-0.0066 - 0.0648)	(0.002 - 0.0713)	(-0.0089 - 0.0751)	(-0.0128 - 0.0765)
Mother secondary	0.0057	0.0127	0.0178**	0.0168	0.0149
	(-0.0116 - 0.0229)	(-0.0031 - 0.0285)	(0.0017 - 0.0338)	(-0.0187 - 0.0523)	(-0.0237 - 0.0535)
Wealth index	-4.48e-08	-3.04e-08	-8.22e-08	-1.85e-07*	-1.85e-07*
	(-4.03e-07 - 3.13e-07)	(-3.69e-07 - 3.08e-07)	(-4.07e-07 - 2.43e-07)	(-3.88e-07 - 1.79e-08)	(-3.93e-07 - 2.22e-08)
Female headed HH	-0.0079	-0.0089	-0.0042	-0.0171	-0.0170
	(-0.0505 - 0.0346)	(-0.0535 - 0.0357)	(-0.0409 - 0.0325)	(-0.0455 - 0.0113)	(-0.0457 - 0.0117)
HH size	-0.0067**	-0.0069**	-0.0071***	-0.0067***	-0.0067***
	(-0.0125 - -0.0010)	(-0.0129 - -0.001)	(-0.0121 - -0.002)	(-0.0119 - -0.0016)	(-0.0117 - -0.0018)
Malaria exposure	0.0023	0.002	0.005	0.004*	0.004
	(-0.0058 - 0.0105)	(-0.0085 - 0.0129)	(-0.0044 - 0.0147)	(-0.0006 - 0.0087)	(-0.0011 - 0.0092)
Rain 25y (dev)	-0.0006	-0.0005	-0.0021	0.0036	0.0036
	(-0.0636 - 0.0625)	(-0.180 - 0.179)	(-0.0293 - 0.0251)	(-0.0076 - 0.0149)	(-0.0086 - 0.0158)
Temp 25y (dev)	-0.0002	-0.0024	-6.14e-05	0.0039	0.0025
	(-0.0106 - 0.0101)	(-0.0374 - 0.0327)	(-0.0041 - 0.0039)	(-0.008 - 0.0158)	(-0.0079 - 0.0129)
Kleibergen-Paap rk Wald F	23.33	22.59	20.46	21.80	26.47
Linear trend		✓			✓
Includes nonresidents			✓	✓	✓
Includes DHS 2013				✓	✓
District & month-year FE	✓	✓	✓	✓	✓
N	4,409	4,409	5,235	11,715	11,715

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1; correction for sampling weights; standard errors clustered at the district level and confidence intervals produced by wild bootstrap (Cameron et al. 2011); full set of controls in Panel B.

Table 7: Regressions of conflict on infant mortality mother fixed effects

Dependent variable:	Mortality rate at 12 months					
	All		Boys		Girls	
	OLS	2SLS	OLS	2SLS	OLS	2SLS
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A:						
Conflict events 12	-0.0012	0.0007	-0.0017***	-0.0035	-0.0007	0.0059*
	(-0.003 - 0.000)	(-0.0046 - 0.0061)	(-0.0029 - -0.0006)	(-0.0100 - 0.003)	(-0.0028 - 0.0014)	(-0.0005 - 0.0124)
Kleibergen-Paap rk Wald F		51.23		31.47		50.47
Panel B:						
Conflict events 12	-0.0011	0.0014	-0.0014***	-0.0026	-0.0006	0.0061*
	(-0.0024 - 0.0003)	(-0.005 - 0.0079)	(-0.0023 - -0.0005)	(-0.0098 - 0.0045)	(-0.0027 - 0.0015)	(-0.0010 - 0.0132)
Twin	0.115*	0.115*	0.110	0.111	0.139	0.141
	(-0.00577 - 0.235)	(-0.00391 - 0.235)	(-0.0242 - 0.245)	(-0.0341 - 0.255)	(-0.132 - 0.410)	(-0.133 - 0.414)
1st child	-0.00534	-0.00628	-0.000959	0.000167	-0.0236	-0.0252
	(-0.0647 - 0.0541)	(-0.0610 - 0.0484)	(-0.0268 - 0.0249)	(-0.130 - 0.131)	(-0.0873 - 0.0401)	(-0.0890 - 0.0387)
# brothers	0.0701***	0.0723***	0.0980***	0.0969***	0.0194*	0.0258**
	(0.0701 - 0.0701)	(0.0723 - 0.0723)	(0.0980 - 0.0980)	(0.0969 - 0.0969)	(-0.00128 - 0.0400)	(0.00278 - 0.0487)
# sisters	0.0469***	0.0470***	-0.00130	-0.00203	0.0645***	0.0650***
	(0.0469 - 0.0469)	(0.0470 - 0.0470)	(-0.0475 - 0.0449)	(-0.0197 - 0.0156)	(0.0645 - 0.0645)	(0.0650 - 0.0650)
Malaria index	0.00692*	0.00364	0.00359	0.00543	0.00270	-0.00678
	(-1.41e-10 - 0.0138)	(-0.00432 - 0.0116)	(-0.00848 - 0.0157)	(-0.00854 - 0.0194)	(-0.00625 - 0.0117)	(-0.0174 - 0.00387)
Rain 25y	-0.00435	-0.00635	-0.0136*	-0.0132	0.00265	-0.00203
	(-0.0236 - 0.0149)	(-0.0270 - 0.0143)	(-0.0287 - 0.00152)	(-0.0313 - 0.00487)	(-0.0312 - 0.0365)	(-0.0288 - 0.0248)
Temp 25y	0.00726	0.00749	0.0121	0.0127	0.00224	0.00191
	(-0.0112 - 0.0258)	(-0.0108 - 0.0258)	(-0.0137 - 0.0378)	(-0.0108 - 0.0361)	(-0.0246 - 0.0291)	(-0.0229 - 0.0267)
Kleibergen-Paap rk Wald F		54.84		34.47		51.33
Mother FE	✓	✓	✓	✓	✓	✓
Month-year FE	✓	✓	✓	✓	✓	✓
N	8,869	8,869	4,460	4,460	4,409	4,409

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1; correction for sampling weights; sample of residents only; Standard errors clustered at the district level and confidence intervals produced by wild bootstrap (percentile-t method).

Table 8: Alternative IV specifications with mother fixed effects

Dependent variable:	Girls' mortality at 12 months				
	2SLS	2SLS	2SLS	2SLS	2SLS
	(1)	(2)	(3)	(4)	(5)
Panel A: No controls					
Conflict events 12	0.0059*	0.0057	0.0052*	0.0036***	0.0034***
	(-0.0005 - 0.0124)	(-0.0011 - 0.0126)	(-0.0003 - 0.0107)	(0.0013 - 0.0059)	(0.0013 - 0.0055)
Kleibergen-Paap rk Wald F	51.23	30.25	50.47	48.51	112.7
Panel B: Full set of controls					
Conflict events 12	0.0061*	0.0057	0.0055*	0.0036***	0.0033***
	(-0.0010 - 0.0132)	(-0.0013 - 0.0128)	(-0.0005 - 0.0115)	(0.0036 - 0.0036)	(0.0016 - 0.0048)
Twin	0.141	0.142	0.110	0.106*	0.106**
	(-0.133 - 0.414)	(-0.133 - 0.418)	(-0.126 - 0.346)	(-0.0036 - 0.216)	(-0.0263 - 0.1895)
1st child	-0.0252	-0.0224	-0.0349	-0.00973	-0.0089
	(-0.0890 - 0.0387)	(-0.0857 - 0.0409)	(-0.0870 - 0.0173)	(-0.0365 - 0.0170)	(-0.0338 - 0.0170)
# brothers	0.0258**	0.0218	0.0224**	-4.94e-05	-0.0047
	(0.0028 - 0.0487)	(-0.0043 - 0.0479)	(0.0045 - 0.0403)	(-0.0014 - 0.0013)	(-0.0176 - 0.0074)
# sisters	0.0650***	0.0701***	0.0659***	0.0616***	0.065
	(0.0650 - 0.0650)	(0.0701 - 0.0701)	(0.0659 - 0.0659)	(0.0616 - 0.0616)	(0.0510 - 0.0797)
Malaria index	-0.0068	-0.0063	-0.0054	0.0039	0.0048
	(-0.0174 - 0.0039)	(-0.0173 - 0.0046)	(-0.0139 - 0.003)	(-0.0085 - 0.0165)	(-0.0053 - 0.0145)
Rain 25y	-0.002	-0.0007	-0.0023	6.63e-05	0.0003
	(-0.0288 - 0.0248)	(-0.0180 - 0.0167)	(-0.0247 - 0.0201)	(-0.0518 - 0.0519)	(-0.0081 - 0.0096)
Temp 25y	0.0019	0.0009	0.0017	0.0058	0.0055
	(-0.0229 - 0.0267)	(-0.0181 - 0.0199)	(-0.0162 - 0.0197)	(-0.00763 - 0.0193)	(-0.0059 - 0.0166)
Kleibergen-Paap rk Wald F	54.84	33.08	51.33	49.31	109.1
Linear trend		✓			✓
Includes nonresidents			✓	✓	✓
Includes DHS 2013				✓	✓
Mother & month-year FE	✓	✓	✓	✓	✓
N	4,409	4,409	5,235	11,715	11,715

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1; correction for sampling weights; standard errors clustered at the district level and confidence intervals produced by wild bootstrap (Cameron et al. 2011); full set of controls in Panel B.

Table 9: Panel regressions on the impact of conflict on child mortality

Dependent variable:	Mortality rate at 12 months					
	All		Boys		Girls	
	OLS	2SLS	OLS	2SLS	OLS	2SLS
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: District, month-year FE						
Conflict events 12	-0.0010**	0.0028	-0.0009*	-0.0009	-0.0002	0.0059**
	(0.0005)	(0.0029)	(0.0005)	(0.0033)	(0.0006)	(0.0024)
N	2,498	2,498	2,498	2,498	2,498	2,498
Kleibergen-Paap rk Wald F		61.96		61.96		61.96
Panel B: District, month-year FE, all controls						
Conflict events 12	-0.0010**	0.0030	-0.0010*	-0.0001	-0.0002	0.0052**
	(0.0005)	(0.0027)	(0.0005)	(0.0031)	(0.0006)	(0.0022)
N	2,498	2,498	2,498	2,498	2,498	2,498
Kleibergen-Paap rk Wald F		71.64		71.64		71.64

Notes: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$; sample of residents only; standard errors clustered at the district level; full set of controls in Panels B.

Table 10: IV regressions of conflict on girl mortality - behavioral factors

Dependent variable:	Girls' mortality at 12 months				
	(1)	(2)	(3)	(4)	(5)
Panel A: Female headed HH					
Conflict Events	0.0051**	0.0045*	0.0037**	0.0029***	0.0025***
	(0.0026)	(0.0026)	(0.0017)	(0.0010)	(0.0010)
Conflict*Female headed HH	-0.0039	-0.0036	-0.0042	-0.0024	-0.0024
	(0.0039)	(0.0038)	(0.0040)	(0.0019)	(0.0019)
Kleibergen-Paap rk Wald F	9.248	8.169	7.214	8.754	9.060
N	4,409	4,409	5,235	11,715	11,715
Panel B: Widow					
Conflict Events	0.0045*	0.0039	0.0033**	0.0026***	0.0022**
	(0.0026)	(0.0026)	(0.0017)	(0.0010)	(0.0009)
Conflict*Widow	0.0014	0.0015	0.0010	-0.0003	-0.0002
	(0.0043)	(0.0042)	(0.0038)	(0.0021)	(0.0022)
Kleibergen-Paap rk Wald F	12.57	11.83	10.23	7.072	6.195
N	4,409	4,409	5,235	11,715	11,715
Full set of controls	✓	✓	✓	✓	✓
Linear trend		✓			✓
Includes nonresidents			✓	✓	✓
Includes DHS 2013				✓	✓
District FE	✓	✓	✓	✓	✓
Month-year FE	✓	✓	✓	✓	✓

Notes: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$; correction for sampling weights; sample of residents only; Standard errors clustered at the district level.

Table (. . .) : IV regressions of conflict on girl mortality - behavioral factors

Dependent variable:	Girls' mortality at 12 months				
	(1)	(2)	(3)	(4)	(5)
Panel C: Number of brothers					
Conflict Events	0.0045*	0.0044	0.0031	0.0027**	0.0024**
	(0.0026)	(0.0028)	(0.0019)	(0.0012)	(0.0011)
Conflict* # brothers	0.0002	-0.0003	0.0004	-0.0001	-0.0002
	(0.0010)	(0.0010)	(0.0009)	(0.0004)	(0.0004)
Kleibergen-Paap rk Wald F	11.50	9.738	7.980	8.778	9.252
N	4,409	4,409	5,235	11,715	11,715
Panel D: Number of sisters					
Conflict Events	0.0059*	0.0057*	0.0041*	0.0033**	0.0029**
	(0.0031)	(0.0033)	(0.0021)	(0.0015)	(0.0014)
Conflict* # sisters	-0.0011	-0.0014	-0.0007	-0.0007	-0.0006
	(0.0010)	(0.0010)	(0.0008)	(0.0006)	(0.0006)
Kleibergen-Paap rk Wald F	8.242	6.740	6.273	7.358	7.744
N	4,409	4,409	5,235	11,715	11,715
Panel E: Son preference					
Conflict Events	0.0054**	0.0047*	0.0041**	0.0029**	0.0025*
	(0.0027)	(0.0028)	(0.0020)	(0.0014)	(0.0013)
Conflict*Son preference	-0.0004	-0.0005	-0.0004	-0.0001	-0.0001
	(0.0007)	(0.0008)	(0.0005)	(0.0004)	(0.0004)
Kleibergen-Paap rk Wald F	17.30	15.11	12.76	12.26	12.67
N	4,038	4,038	4,782	10,637	10,637
Full set of controls	✓	✓	✓	✓	✓
Linear trend		✓			✓
Includes nonresidents			✓	✓	✓
Includes DHS 2013				✓	✓
District FE	✓	✓	✓	✓	✓
Month-year FE	✓	✓	✓	✓	✓

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1; correction for sampling weights; sample of residents only; Standard errors clustered at the district level.

Table 11: Regressions of conflict on ex ratio (boys/total births) (2SLS) - biological factors

	Conflict events 12m		Conflict exposure 12m	
	[1]	[2]	[3]	[4]
Panel A: Sex ratio at birth				
Conflict in utero	-0.0068 *	-0.0071*	-0.0540	-0.0553*
	(0.0041)	(0.0041)	(0.0341)	(0.0331)
F-stat of IV	26.07***	32.99***	43.16***	49.56***
N	2,598	2,598	2,598	2,598
Panel B: Sex ratio at 12 months—impact in utero				
Conflict in utero	-0.0055	-0.0062	-0.0442	-0.0484
	(0.0043)	(0.0043)	(0.0351)	(0.0345)
F-stat of IV	26.07***	32.99***	43.16***	49.56***
N	2,598	2,598	2,598	2,598
Panel C: Sex ratio at 12 months—separate impacts in utero and first year				
Conflict in utero	-0.0058	-0.0068	-0.0444	-0.0455
	(0.0063)	(0.0061)	(0.0343)	(0.0301)
Conflict	0.0007	0.0019	-0.0014	0.0045
	(0.0074)	(0.0073)	(0.0362)	(0.0364)
F-stat of IV (in utero)	29.37***	20.87***	21.20***	30.54***
F-stat of IV (Conflict)	13.84***	35.25***	20.85***	27.71***
N	2,598	2,598	2,598	2,598
Panel D: Sex ratio at 12 months—total impact in utero and first year				
Conflict in utero & 12 months	-0.0027	-0.0024	-0.0253	-0.02164
	(0.0024)	(0.0022)	(0.0225)	(0.0199)
F-Stat of IV	44.17***	65.41***	27.60***	43.21***
N	2,598	2,598	2,598	2,598
Linear trend		✓		✓
District & month-year FE	✓	✓	✓	✓

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1; correction for sampling weights; sample of residents only; standard errors clustered at the district level. 2SLS = two-stage least squares; FE = fixed effects; IV = instrumental variables.

Figure 1: Map of conflict events in Democratic Republic of Congo, 1997–2004

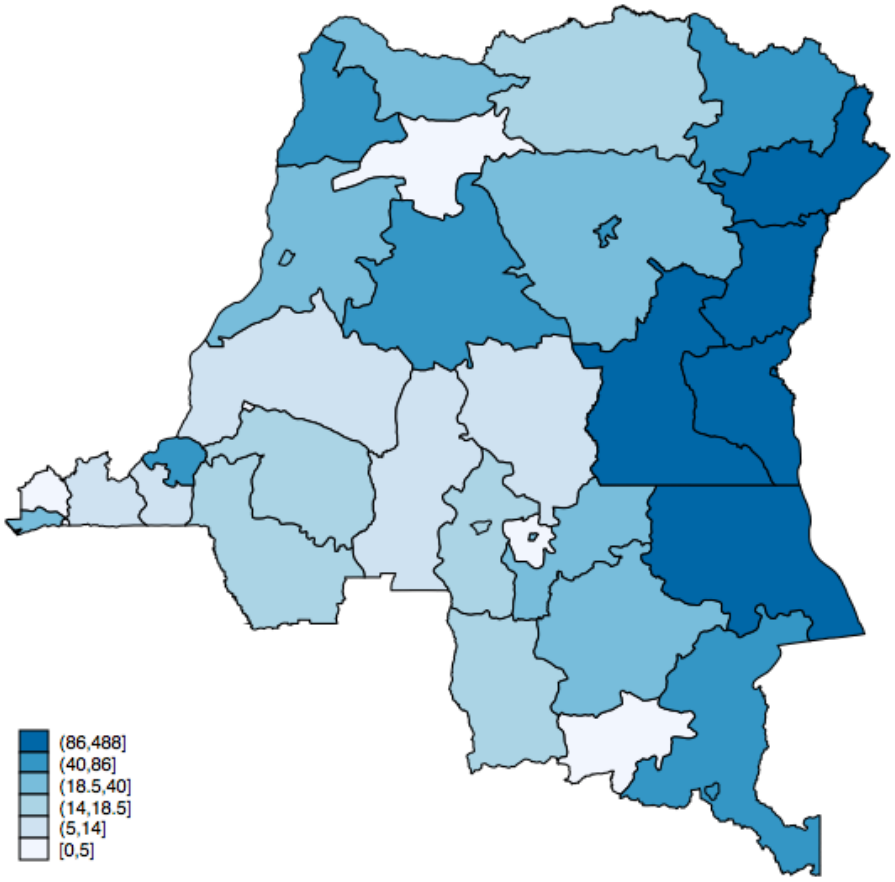

Figure 2: Map of mineral deposits in Democratic Republic of Congo

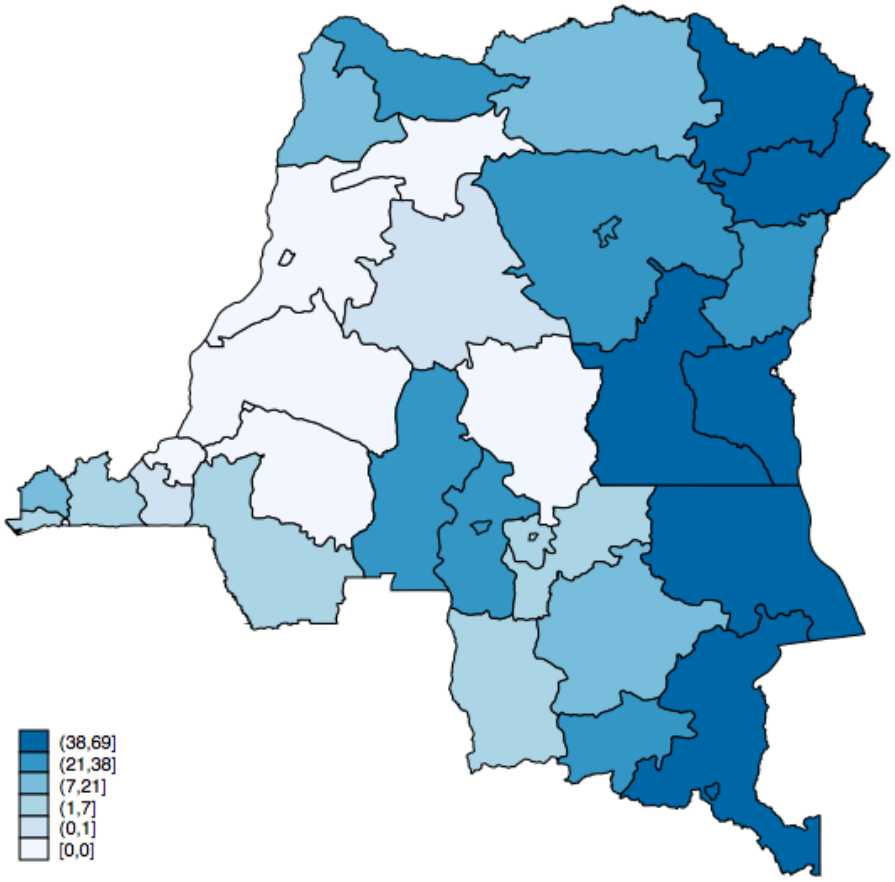

Figure 3: Lowess estimation of first stage

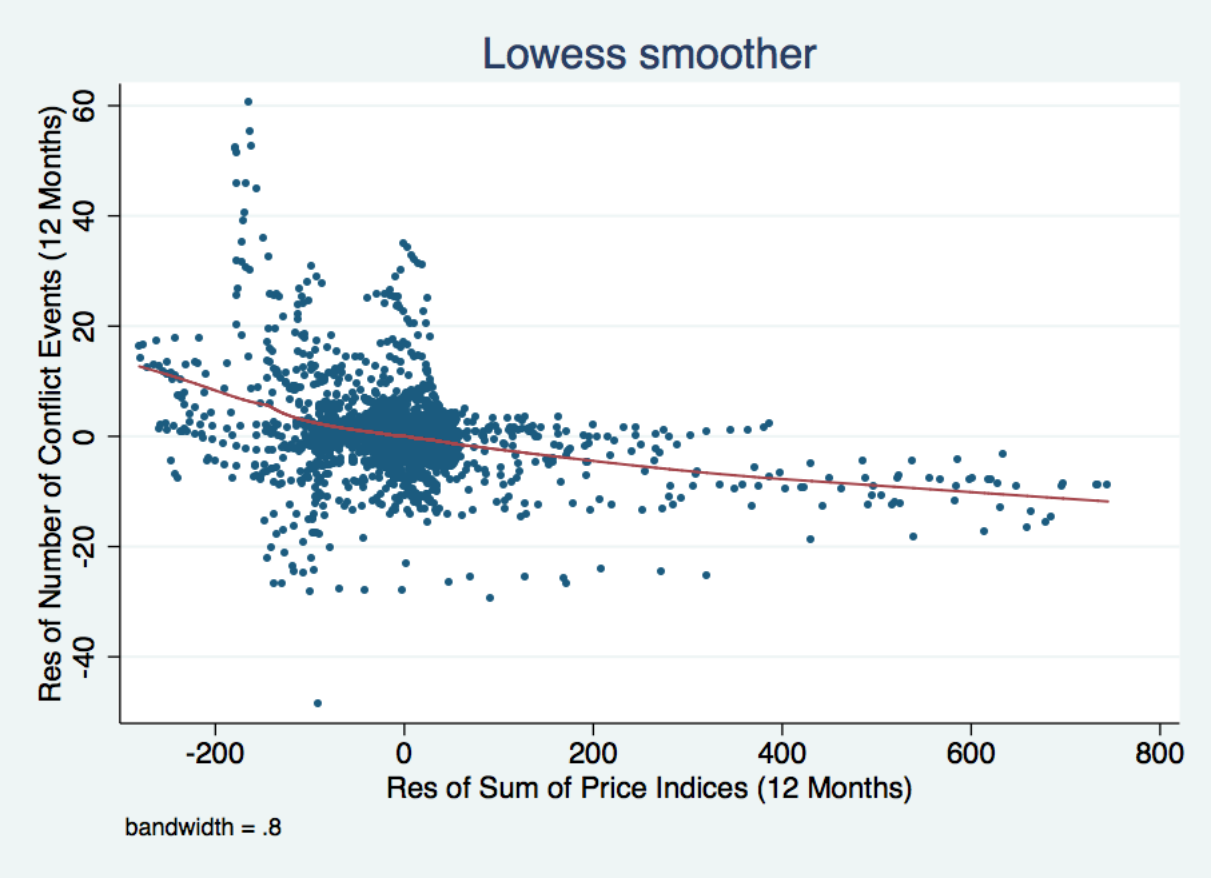

Table A.1: Cross sectional regressions : Alternative Conflict

Dependent variable:	Girls' mortality at 12 months					
	Excluding DHS 2013			Including DHS 2013		
	2SLS	2SLS	2SLS	2SLS	2SLS	2SLS
	(1)	(2)	(3)	(4)	(5)	(5)
Panel A: No controls						
Conflict exposure	0.0498*			0.0227		
	(-0.001 - 0.100)			(-0.005 - 0.051)		
UCDP events		0.0924*			0.0462	
		(-0.002 - 0.186)			(-0.011 - 0.103)	
UCDP fatalities			0.0009*			0.0005
			(-0.000 - 0.002)			(-0.000 - 0.001)
Kleibergen-Paap rk Wald F	11.55	0.804	3.215	14.50	1.109	3.215
Panel B: Full set of controls						
Conflict exposure	0.0572**			0.0276*		
	(0.0043 - 0.110)			(-0.0014 - 0.0565)		
UCDP events		0.0955**			0.0497*	
		(0.00722 - 0.184)			(-0.00249 - 0.102)	
UCDP fatalities			0.0012**			0.0007*
			(9.07e-05 - 0.0023)			(-3.43e-05 - 0.0014)
Kleibergen-Paap rk Wald F	10.87	1.301	2.087	12.72	1.431	2.087
Includes DHS 2013				✓	✓	✓
District & month-year FE	✓	✓	✓	✓	✓	✓
N	4,409	4,409	4,409	11,715	11,715	11,715

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1; correction for sampling weights; standard errors clustered at the district level and confidence intervals produced by wild bootstrap (Cameron et al. 2011); full set of controls in Panel B.

Figure A.1: Placebo test on girls sample