HEDG
HEALTH, ECONOMETRICS AND DATA GROUP
THE UNIVERSITY of York

WP 12/11

Children of the Revolution: Fetal and Child Health amidst Violent Civil Conflict

Christine Valente

July 2011

york.ac.uk/res/herc/hedgwp

Children of the Revolution: Fetal and Child Health amidst Violent Civil Conflict

Christine Valente*
University of Sheffield, U.K.

July 6, 2011

Abstract

This paper considers the impact of exposure to violent conflict *in utero*, and after birth, on a range of fetal and child health inputs and outcomes, using the Maoist insurgency that affected Nepal between 1996 and 2006 as a case study. Conflict intensity is measured by the number of conflict deaths by district and month and merged with pregnancy histories from the 2001 and 2006 Demographic and Health Surveys. Maternal fixed-effects estimation allows me to control for-, and shed light on- selection into becoming pregnant and giving birth at times of more intense conflict. Exposure to conflict in the first few years of life has an adverse effect on child nutritional status. However, exposure to conflict *in utero* has both scarring and selection effects on survivors. As conflict intensity increases, the likelihood of miscarriage increases, and so a smaller share of the frailer fetuses is carried to term. This selection effect tends to dominate in the second trimester of pregnancy, whilst scarring effects are stronger in the third trimester. Use of health care such as antenatal care, (medical) help with delivery, and immunization do not appear to decrease when conflict intensifies, and there is no evidence of acute maternal malnutrition, thus suggesting a role for other factors such as psychological stress in the increased probability of miscarriage.

JEL codes: I10, J13, O15
Keywords: civil conflict, child health, fetal loss, Nepal

*Email address: christine.valente@sheffield.ac.uk. I thank the Informal Sector Service Centre (INSEC) for granting me access to their conflict database. I also thank Andy Dickerson, Arne Risa Hole, Maarten Lindeboom, Grant Miller, Robert Scholte and participants at the International Workshop on the Global Costs of Conflict in Berlin, at the DIAL Development Conference in Paris, and seminars at the Tinbergen Institute, and at the University of Leeds, Nottingham, and Sheffield for their useful comments. All errors are my own.

1 Introduction

More than half of all countries have experienced at least one episode of civil conflict since 1950 and a third have gone through full-fledged civil *war*. However, the body of knowledge on the causes and consequences of conflict is still limited and disputed (Blattman & Miguel 2010).

There is burgeoning microeconometric evidence of an adverse impact of violent conflict on health, and on child health in particular (Alderman et al. (2006); Akresh et al. (2009); Akbulut-Yuksel (2009); Akresh et al. (2010)). Health before birth and in early life is not only intrinsically valuable, it also has persistent effects into adulthood, and is instrumental in the accumulation of education (see Strauss & Thomas (2008) and Glewwe & Miguel (2008) for recent surveys). Countries affected by civil war may therefore bear the cost of war long after the end of the fighting if child health is adversely affected. One question which has received little attention so far is that of selection on fetal health at times of war. The biomedical literature suggests a number of pathways for violent conflict to translate into poorer child and adult health (i.e., a scarring effect), but also mechanisms leading to increased rates of fetal loss, and therefore "positive" selection into birth on fetal health. As a consequence, the effect of exposure to conflict *in utero* could be to shift either downwards or upwards the distribution of the health endowment amongst children effectively born.

Fetal loss is by no means a negligible phenomenon: in clinical studies in developed countries, 12-15 percent of clinical pregnancies (i.e., more than 6 weeks after the last menstrual period) end in a miscarriage, much more if pre-clinical pregnancies are included, whilst typically less than one percent end in a still birth (Garcia-Enguidanos et al. (2002); Cnattingius et al. (1998); Cai & Feng (2005); Nepomnaschy et al. (2006)). The possibility of increased fetal loss during times of civil conflict and war has been suggested before (e.g., Wynn & Wynn (1993); Rajab et al. (2000); Roseboom et al. (2001)), and previous research has found that psychological stress and maternal malnutrition can increase both fetal loss and adverse birth outcomes for children who are born alive (Herrmann et al. (2001); Siega-Riz et al. (2001); Mulder et al. (2002); Cai & Feng (2005); Helgstrand & Andersen (2005); Nepomnaschy et al. (2006); Maconochie et al. (2007); Wisborg et al. (2008)). But to the best of my knowledge this is the first study showing direct, arguably causal, evidence of the effect of conflict on fetal loss.[1]

The case of Nepal provides a particularly useful case study. It raises an interesting puzzle since child health indicators such as child mortality and immunization rates have improved steadily over the conflict period (1996-2006) (World Bank (2005); MOHP, New Era and Macro International Inc. (2007); Macours (2011)). The conflict was also of moderate intensity, and so extends our understanding of the impact of violent conflict along the distribution of conflict intensity.[2] Partly due to the relatively low intensity of this conflict, and thanks to the efforts of a civil rights NGO with representatives in all 75 districts of Nepal, the Informal Sector Service Center (INSEC), exceptionally detailed conflict intensity data are available, namely deaths counts per district and month over the whole conflict period. Similarly, one of the major limitations of studies investigating the impact of conflict, namely conflict-induced migration, is minimized in the case of Nepal

[1] See Appendix Section A-2 for a detailed review of the existing biomedical literature.

[2] The case studies analyzed in previous research tend to focus on conflicts of extremely high intensity, such as the bombing of Germany by allied forces during WWII, the recent civil war in Burundi, where 200,000 people died (Akresh et al. 2009), and the Ethiopia-Eritrea war, where between 70,000 and 100,000 individuals lost their lives (Akresh et al. 2010).

where less than one percent of the population is estimated to have been displaced by the conflict (US Agency for International Development 2007). Last but not least, rare data on pregnancies not ending in a live birth are available from the 2001 and 2006 Demographic and Health Surveys of Nepal for the entire conflict period, so that it is possible to study the impact of conflict exposure *in utero* on pregnancy resolution (live birth, miscarriage, still birth).

A related, and growing body of research is concerned with understanding the extent to which insults to maternal health are transmitted through to their offspring, whether they have occurred over the course of the mother's life before conception, or during the course of gestation (Barker (1995); Almond (2006); Meng & Qian (2009)). The influential "fetal programming" hypothesis, which originally links fetal undernutrition in middle to late gestation to later coronary heart disease (Barker 1995) has paved the way to a number of studies of different pathways through which an unhealthy fetal environment during gestation - and famine in particular - can affect health and socioeconomic outcomes all the way to adult life (Barker (1995); Roseboom et al. (2001); Painter et al. (2005); Almond et al. (2010); Meng & Qian (2009); Almond & Currie (forthcoming)).

Another strand of literature which the present analysis speaks to is that concerned with the effects of economic shocks on fetal and child health, which have been found to be either pro-cyclical or counter-cyclical, depending on the population and economic shock analyzed (Dehejia & Lleras-Muney (2004); Miller & Urdinola (2010)). The concerns of this literature can be readily extended to civil conflict: if different types of mothers are more or less likely to postpone or experience difficulties in child bearing during periods of political instability, then this should influence the health of children conceived and born at times of civil strife, in a direction that is undetermined *a priori*. And if fetal and child health inputs are time-intensive, then a slowing down of the economic activity due to conflict may have a positive effect on fetal and child health, which could compensate for the loss of income.

This paper provides further evidence of the impact of exposure to civil conflict on child nutritional status. It extends the analysis of the impact of conflict on a range of health inputs and outcomes, from conception to age five, and paying particular attention to selection processes and to differentiating precisely between the effect of exposure of the child whilst *in utero* and exposure after birth. The key contribution of this paper is that selection on fetal health is analyzed directly by estimating the impact of exposure to conflict on fetal loss.

I find evidence of (adverse) scarring effects of exposure to conflict on child nutritional status, as well as of positive selection effects due to fetal loss. Whilst any scarring effect dominates for *in utero* exposure during the third trimester of pregnancy, and exposure after birth, selection appears to dominate for second trimester exposure, and the total effect of first trimester *in utero* exposure is not significantly different from zero. There is selection both on observed- and unobserved parental characteristics, which, when not controlled for, leads to the underestimation of the effect of conflict on fetal and child health. Findings are robust to a number of checks including the inclusion of district-specific trends and placebo experiments.

Section 3 depicts the conflict background, Section 4 presents the data and identification strategy, Section 5 reports the results, Section 6 checks the robustness of these findings, Section 7 explores underlying mechanisms, and Section 8 concludes.

2 Civil Conflict in Nepal

Nepal became a parliamentary monarchy in 1990. Despite multiparty democratic elections being then held in Nepal for the first time, a Maoist insurgency broke out in 1996, only to end in 2006. The insurgency started in February 1996 in the Rolpa district. At first, it was concentrated in a few Communist strongholds in Western Nepal, but by the end of the war, conflict-related casualties were recorded in 73 out of the 75 Nepalese districts. The Maoist presence varied from sporadic attacks to total control where they organized their own local governments and law courts. Over the course of the conflict, Maoists attacked government targets such as army barracks, police posts, local government buildings (Do & Iyer forthcoming). They were also reported to terrorize, loot, abduct, and physically assault civilians (Human Rights Watch (2004); Bohara et al. (2006)). On the other hand, government security forces also killed civilians and were accused of using children for spying, torturing, displacing and summarily convicting civilians (Bohara et al., 2006).

A crucial moment in the conflict was the end put in November 2001 by the Maoists to a short-lived cease-fire agreed earlier that year. From then on, the government's response intensified dramatically, involving the Royal Nepal Army, leading to an escalation of violence (see Figure 1). Building on opposition to King Gyanendra's authoritative reaction to the prolonged conflict, the Maoists joined forces with some of the country's major political parties, leading to the signature of a peace agreement in November 2006 and the creation of an interim government led by a power-sharing coalition including the Maoists. This put an end to a 10-year long conflict that led to the deaths of over 13,000 people and the displacement of an estimated 200,000 (US Agency for International Development 2007).

The intensity of conflict varied much across districts of Nepal, as illustrated in Figure 2, which depicts the distribution of districts between three groups of 25 districts each, defined by terciles of the distribution of conflict deaths per 1000 inhabitants.

Several arguments have been put forward to explain the district variation in the intensity of the insurgency, including geography (Murshed and Gates, 2005; Bohara et al. (2006); Do & Iyer (forthcoming)), poverty (Murshed & Gates (2005); Do & Iyer (forthcoming)), lack of political participation (Bohara et al. 2006), and inter-group inequality (Murshed & Gates (2005); Macours (2011)). Given that these variables are likely to also affect fetal and child health, it is important to control for such variables in the analysis, as discussed in the next section.

One would generally expect exposure to conflict to be detrimental to maternal and infant health, through the direct effect of violence as well as its indirect effect on maternal nutrition and stress, disease prevalence, investments in (health) human capital in a context of altered expectations, and access to health care, either through destruction or lower income at the local level due to war-related disruption and looting. However, the predicted effect of the Nepalese conflict on maternal and child health is less clear-cut than in most violent conflict instances. Indeed, the impact of the conflict on the overall economy is unclear, child survival improved dramatically at the national level, and education was seemingly little affected (World Bank (2005); Macours (2011); Valente (2011)). In addition, most commentators agree that, although Maoist insurgents are said to have destroyed more than 1000 rural health posts as collateral damage to the destruction of other government buildings, and of having harassed, racketed, and stolen drugs from health workers, they also appear to have shown some commitment to their stated policy of avoiding disruption of "people's services", and are even said to have helped promote ini-

tiatives such as the national immunization programs and vitamin A supplementation and de-worming campaigns, contributed to reduced health staff absenteeism, and set-up their own health services in remote areas under their control (e.g., Beun & Neupane (2003); Devkota (2005); Collins (2006)).

3 Data and Identification Strategy

3.1 Data

Demographic and Health Surveys (DHS) have been carried out in a number of developing countries as part of the Measure DHS project, a worldwide USAID-funded project aimed essentially at providing detailed, reliable information on fertility, family planning, maternal and child health and mortality.

The second and third DHS carried out in Nepal took place in 2001 and 2006, respectively. These collected data from a nationally representative sample of women aged between 15 and 49 (if ever married in the case of the 2001 survey). Respondents were asked about their entire fertility history, including dates of all births and deaths of any liveborn child and dates of end and duration of all other pregnancies.[3] The questionnaires contain a number of probes for these, and enumerators are specifically trained to ensure that this information, that is central to the survey, is reliable.[4] These fertility histories are used here to create a panel dataset where mothers are the cross-sectional units and pregnancies the "longitudinal" unit, as in Bhalotra & van Soest (2008).

Due to the retrospective nature of the data, there may be measurement error in the dependent variable. Beckett et al. (2001) find that recall error in fertility histories is not an issue for live born children, except for some age heaping (e.g., rounding at one year old for children who die when 11 or 13 months old). As a consequence, I allow for age heaping such that the neonatal mortality indicator switches on for children who were reported to be up to one month old at the age of death.[5] I also address this issue by restricting the analysis to children born no longer than 5 years before the start of the survey.[6]

Data on pregnancies that do not result in a live birth are prone to more measurement error, especially in the form of underreporting (Beckett et al. 2001). By restricting the sample to the five years preceding each survey, underreporting should be as low as possible based on survey data. It is important to impose this stringent recall cut-off in the present analysis because conflict intensity varies substantially from month to month (see Figure 1), and so it is important to minimize measurement error in the dates of start and end of pregnancy.

Different women were interviewed in the 2001 and 2006 DHS surveys. However, the degree of measurement error in the reporting of neonatal deaths and miscarriages can be appraised by comparing the average rates of each of these outcomes obtained for children conceived in a given (Nepali) calendar year, but reported by different mothers 5 years apart (in 2001 and in 2006), as depicted by Figures 3 and 4. These graphs show that, for

[3] In these surveys, women were asked to report each of their pregnancies in turn, and, one by one, whether the baby was "born alive, born dead, or lost before birth". If they answered either of the two last options, the respondents were then asked about the month and year the pregnancy ended and its duration. If the child was born alive, I set the duration of the pregnancy to be 9 months.

[4] See MOHP, New Era and Macro International Inc. (2007) for more information.

[5] Strictly speaking, neonatal mortality relates to mortality in the first 4 weeks of life.

[6] In the case of pregnancies that do not end in a live birth, the sample is restricted to pregnancies starting no longer than 5 years and 9 months before the start of the survey.

the recent period covered by the data used in this paper, average neonatal mortality and miscarriage rates are reasonably consistent across surveys, especially considering that the rates are based on comparatively small year samples and on different women. It is also worth noting the sharp increase in miscarriages coinciding with the conflict escalation, but the absence of such movement in neonatal mortality.

In addition to the entire fertility histories of all interviewed women, for the subsample of children born up to five years before the survey, the DHS also collected more detailed information on prenatal care, delivery, immunization status, and children height and weight (measured by enumerators using internationally recognized instruments). By pooling the 2001 and 2006 DHS cross-sections, I obtain a sample of children aged five or below at the time of either survey whose dates of birth span the whole period of the conflict, namely 1996-2006.

The individual data from the pooled Nepalese DHS are then merged with the number of conflict deaths per month per district of Nepal compiled by the Informal Sector Service Center (INSEC). All conflict exposure variables are expressed in deaths per 1000 district inhabitants as per the last pre-conflict census (1991).

The DHS surveys took place either before or after the conflict escalation, so that data collection was not much disrupted. In 2001, six out of 257 sampling units had to be dropped from the sample for security reasons (MOHP, New Era and ORC Macro (2002), p.6). But in 2006, conflict was nearly over everywhere, and so data collection could proceed without hindrance.

I restrict the analysis to singletons, as is standard in the demographic literature, since including multiple births can bias estimates. I also restrict the analysis to children who were conceived in the place where their mothers were interviewed in order to limit measurement error in exposure to conflict.[7]. When analyzing the impact of violent conflict on pregnancy resolution, I drop pregnancies starting less than 9 months before the date of interview since, for this time period, only pregnancies that do not end in a live birth are recorded in the data, and drop pregnancies that do not end in a live birth but for which the information on duration of pregnancy is missing (less than 1 percent of pregnancies not ending in a live birth). The resulting pregnancies sample size is 12,490. After dropping pregnancies that did not end in a live birth (1074), children who were not born at least a full month before the interview (244), and those whose mothers did not report their (subjective) size at birth (574), the births sample counts 10,598 children. Among these, prenatal care variables were only collected for the last child born to each mother. Delivery information was collected for all children under 5 years old. Anthropometric data were collected for all children under 5 years old at the time of the survey, with about 3 percent of missing observations (depending on the survey round). Finally, child immunization information was collected for all children under 5 years old who were alive at the time of the survey, but here I restrict the sample to children aged one to five for the immunization analysis because full immunization is normally achieved within 12 months.[8]

A number of health outcomes are considered, namely binary indicators for miscarriage, still birth, size of baby at birth as reported by the mother, neonatal mortality, the standardized score of under-5 children for height-for-age (HAZ), and a binary variable for

[7]This drops 8.7 (8.4) percent of conceptions (pregnancies), but my findings are not sensitive to including these children (see Section 5).

[8]According to WHO guidelines, children are fully immunized when they have received one dose of the tuberculosis vaccine (BCG) and measles vaccine, and three doses of the polio and DPT vaccines. They are normally administered within the first 14 weeks of life except for the measles vaccine which is normally given shortly after the age of 9 months (MOHP, New Era and Macro International Inc. 2007).

stunting, which switches on for children with a height-for-age z-score below 2 standard deviations of the reference population. Other left-hand side variables used in the paper capture different aspects of antenatal and postnatal care, namely: the number of antenatal care visits, a dummy for whether or not the mother received iron and folic tablets during pregnancy, a dummy for whether or not the mother received a tetanus injection during pregnancy, two dummies for the nature of help received during delivery (medical/non-medical or none/some), and two variables capturing immunization status for children at least one year old (one dummy for full immunization, and one for no immunization).

Miscarriages and still births are studied separately. For each pregnancy, mothers are asked whether the baby was "born alive, born dead, or lost before birth". If they answer that the baby was born dead or lost before birth, mothers are then asked whether they or someone else had done "something to end this pregnancy" (MOHP, New Era and Macro International Inc. 2007). The miscarriage (still birth) variable is equal to one if the mother answers that the baby was "lost before birth" ("born dead") without any action taken to end the pregnancy, and zero if the child was born alive. When the mother answered that some action was taken to end the pregnancy, or when she gave no answer about intent, the miscarriage and still birth indicators are set to missing in order to focus on biological mechanisms.

Although newborn health is difficult to capture with a single variable, birth weight is a commonly used measure. In a country like Nepal, where over 80 percent of babies are born at home (MOHP, New Era and Macro International Inc. 2007), birth weight is unknown for the majority of children. However, the DHS asked mothers to report whether at birth the child was 'very large', 'larger than average', 'average', 'smaller than average' or 'very small' at birth. I use this information to create a 'small baby' dummy equal to one if the child was 'smaller than average' or 'very small'.

Child nutritional status is captured by children's height-for-age z-scores (HAZ) and a stunting indicator based on HAZ. These are widely used indicators of long term growth retardation (see the seminal contribution of Martorell & Habicht (1986)). The reference population used to calculate the z-scores is the WHO Child Growth Standards, which is based on healthy children from Brazil, Ghana, India, Norway, Oman and the USA.

Summary statistics for the key variables used in the analysis are reported in Table 1. For the purpose of this table, the sample is divided into three groups, based on the district's position in the distribution of total district deaths (per 1000 inhabitants) over the conflict period. Fertility is lower in the low conflict intensity tercile, as illustrated by the lower proportion of children of pregnancy order five and above in this district group. Children born in the low conflict intensity tercile are more often born in urban areas, and to educated mothers owning more household assets. However, children in the low conflict-intensity tercile are less often born to parents from the more privileged classes (Brahmin and Chhetri) and more likely born to somewhat less well-off other Tarai/Madhesi castes, Janajati, and Muslim parents. They are less likely to be small babies at birth and possibly as a consequence of this are less often small for their age. Their mothers receive more antenatal care and are more likely to receive care from a skilled birth attendant at delivery. However, there is no difference in the probability of neonatal death across conflict-intensity groups, and no clear correlation between conflict intensity and the likelihood of miscarriage or still birth. Children from the lowest conflict intensity tercile are also more often wasted, i.e., have lower weight for height, and do not receive better or worse immunization. It is interesting to remark that the largest difference in conflict intensity is observed between the intermediate and high-intensity terciles, whilst the characteristics of children in these

two groups are very similar, except for miscarriages, which are more common in the high conflict-intensity tercile. Unless otherwise specified, the magnitude of the estimated effects are interpreted in relation to the change in the outcome of interest when going from the mean conflict exposure in the low conflict-intensity tercile to the high conflict-intensity tercile (e.g., in the case of exposure *in utero*, for a change of $0.011 - 0.002 = 0.009$).

3.2 Identification Strategy

I first estimate linear panel data fixed-effects models of the form:

$$Y_{idt} = \beta_0 + \beta_{pre} pre_{dt} + \beta_{utero} inUtero_{dt} [+\beta_{post} post_{dt}] + X'_{idt}\beta_X + Y'_t\beta_y + D_d + u_{idt} \quad (1)$$

where Y_{idt} is one of several pregnancy, mortality, or health outcomes of interest for child i born in district d and conceived in month t; pre_{dt} is the number of (district) conflict deaths that have occurred before the conception of the index child conceived in district d in month t; $inUtero_{dt}$ is the average monthly number of casualties during pregnancy (so as to be comparable between pregnancies with different gestational periods); $post_{dt}$ is the average monthly number of conflict deaths occurring between birth and the interview date, which is included for outcomes realized beyond the neonatal period; X_{idt} are child-specific demographic controls, namely age of mother at conception and its square, pregnancy order indicators (for second, third, fourth, and "five and above"), and 11 calendar month of conception dummies. Y_t is a set of year of conception dummies, D_d a set of district fixed effects, and u_{idt} is an error term assumed to be independent between districts but not necessarily within district, and robust to heteroskedasticity of an arbitrary form.

When Y_{idt} is the nutritional or immunization status of children at the time of interview, I also include a control for age (in months) at measurement. Nutritional status captured by, e.g., height-for-age, is indeed a cumulative variable, and thus tends to deteriorate as the child gets older. It is therefore necessary to control for age at anthropometric measurement where, as is the case here, the treatment variable is correlated with age at measurement (due to the intensification of conflict over time). The same logic applies to immunization status, which is correlated both with conflict intensity and age at interview.[9]

For outcomes that are available for at least two siblings, I also estimate models in which I replace district fixed effects with mother fixed effects, so as to shed light on the degree to which estimates are due to selection of women who become pregnant at times of higher conflict intensity. This is the case for variables that are available over the entire fertile life of respondents, namely whether miscarriage or still birth and neonatal mortality, as well as for some variables collected for *all* children under 5 years old at the time of survey (size at birth, help with delivery, immunization, and height-for-age), but not for antenatal care variables. For clarity, in each mother fixed effects specification, I explicitly restrict the relevant samples to children with at least another sibling in the sample, and refer to this sample as the "siblings" sample.

In an intermediate step between the district fixed-effects analysis and the maternal fixed-effects analysis, I estimate district fixed-effects models including controls for urban residence, maternal education dummies, and caste, in order to see whether controlling for these (largely predetermined) observable maternal characteristics succeeds in accounting

[9] In the within-district specifications, the mechanical relationship between age at interview and exposure to conflict is broken by the fact that children of a given age are observed both in 2001 and 2006. However, in the within-mother specifications, the inclusion of age at interview fixed-effects is particularly important since a given mother is only interviewed in one of the surveys.

for unobserved heterogeneity of parents. I do this both for the whole sample and the siblings sample in order to appraise the singularity of families who have at least two children in the five years preceding the survey.

The estimation framework proposed here follows a difference-in-difference identification strategy, and as such the main threat to identification is the existence of time-varying omitted factors correlated with the health outcomes of interest as well as conflict exposure. Figure 1 shows that casualties vary drastically from one month to the other within district, both increasing and decreasing sharply, so that contemporary changes in omitted determinants of health are less likely to drive the estimates of the effect of conflict exposure *in utero* than in applications relying on cruder measures of conflict intensity.

Nonetheless, several strategies are used in order to address the possibility of time-varying confounders. First, I control for cumulated conflict intensity before conception (pre_{dt}), which should capture pre-conception trends correlated with conflict intensity. Second, I estimate the robustness of my findings to including a linear district-specific trend. Third, I carry out a series of placebo experiments, namely: (i) I test for the presence of pre-conflict differences trends in neonatal and infant mortality and in pregnancy loss (since these variables are defined over the woman's whole fertility history and not only her recent fertility history) for districts with varying degrees of future conflict intensity, as explained in more detail in Section 5 and (ii) I run augmented versions of Equation 1 in which I also include a placebo treatment variable equal to the average monthly number of casualties during the same calendar period as the gestation period, but 12 months earlier. If the estimated effect of conflict exposure *in utero* in Equation 1 were driven by time-varying omitted factors, then the outcome variable should also be correlated with conflict intensity in the previous year. Other tests investigating the robustness of my findings to changes in samples and specification are reported in Section 5.

4 Results

4.1 Selection into Birth at Times of Conflict

4.1.1 Selection on Parental Characteristics

In order to shed light on selection on observable parental characteristics, I estimate variants of Equation 1, in which the dependent variable is, in turn, an indicator for wealth (measured by asset ownership quintile), maternal education, urban location, and caste/ethnicity group. Results presented in Table 2 show that high-caste mothers (Brahmin or Chhetri) are comparatively less likely to become pregnant after they have been exposed to more conflict and when they anticipate more intense conflict during their pregnancy. Similarly, mothers who have some university education are also less likely to become pregnant when they have experienced more conflict or anticipate more conflict during their pregnancy. On the contrary, there is an increase in the proportion of pregnancies from indigenous groups such as the Tarai/Madhesi and, although not quite statistically significantly, the Janajati. This pattern of selection is consistent with the hypothesis that groups who felt most threatened by the Maoist insurgency postponed having children in reaction to the intensity of violence in their area, whilst the reverse applied to groups who supported the insurgency. Namely, going from the mean conflict exposure in the low conflict-intensity tercile to the high conflict-intensity tercile decreases the share of Brahmin or Chhetri pregnancies by 4.2 ppts (combining exposure before and during

the pregnancy), or a decrease of 12 percent, and decreases the likelihood of a pregnancy occurring to a mother with higher-education by 0.6 ppts, or a decrease of 34 percent. A similar movement across the distribution of conflict exposure leads to an increase of 8.3 percent in the proportion of Tarai/Madhesi (0.9 ppts), and of 5.9 percent of Janajati (1.75 ppts). The implications of selection on these observable characteristics for health inputs and outcomes depends on the empirical link between these parental characteristics and particular inputs and outputs. Based on associations in the raw data (Bennett, L. and Ram Dahal, D. and Govindasamy, P. 2008), the expectation is that the decrease in the already very small proportion of highly educated mothers, and of mothers from the highest castes would tend to decrease (worsen) health inputs (outcomes) for children born at times of conflict. This would tend to lead to an overestimation of the adverse effect of conflict when these parental characteristics are not controlled for. In the results to follow, however, this effect is small and more than compensated by selection on unobservable parental characteristics correlated with better health outcomes.

Note also that the existence of selection on observable characteristics of parents is likely to extend to unobservable parental characteristics, and so it is important to account for this selection process when estimating the effect of conflict.

4.1.2 Selection on Fetal Health

Selection on fetal health is investigated in Tables 3 and 4, where I estimate the effect of exposure to violent conflict on the likelihood of miscarriage (Table 3) and still birth (Table 4), against the alternative of a live birth. Each table is arranged as follows. In the first column, I estimate Equation 1 on the whole sample, without controlling for time-invariant maternal characteristics. In the second column, I add controls for urban residence, maternal education dummies, and caste, in order to see whether controlling for these observable maternal characteristics succeeds in accounting for the change in parental composition correlated with conflict intensity. In Column (3), I restrict the sample to pregnancies with at least another sibling in the relevant time period in order to assess whether families with at least two children conceived within 5 years and nine months of the survey are differently affected by violent conflict compared to the rest of the sample. In Column (4) I present maternal fixed effects estimates, which are robust to selection on time-invariant parental heterogeneity. Columns (5) and (6) repeat the regression in Column (4) but allowing for different effects of *in utero* exposure when it takes place at different stages of the pregnancy.

When selection on parental characteristics is not controlled for (Column (1)), exposure to conflict does not appear to have a significant effect on the probability of miscarriage, irrespective of whether I control for observable parental characteristics (Column (2)) or whether I consider the siblings sample only (Column (3)). However, once I allow exposure to conflict to be correlated with unobservable maternal heterogeneity (Column (4)), results indicate that maternal exposure to conflict during pregnancy significantly increases the probability of miscarriage. Going from mean *in utero* exposure in the low-intensity district group to the high-intensity group leads to an increase in the probability of miscarriage by 0.82 ppts (12.9 percent of the mean).

The existing biomedical and economic literature suggests that maternal stress experienced in the first trimester has a larger effect on pre-term birth than shocks experienced later in gestation. In addition, in the current sample 91 percent of miscarriages occur in the first five months (see Table 1), as would be expected. The robustness of the estimated effect of exposure to conflict *in utero* on miscarriage can thus be tested by dividing the

pregnancy period in two phases, the first one starting in the month of conception (mc) and lasting until $mc+4$, and the second spanning $mc+5$ to $mc+9$. The results are presented in Column (5) and confirm that the effect of conflict exposure *in utero* on miscarriage is driven by conflict during the first 5 months of the pregnancy. When trying to ask more of the data by splitting the pregnancy period in three trimesters (mc to $mc+3$, $mc+3$ to $mc+6$, $mc+6$ to $mc+9$), the magnitude of the effect of exposure in the first trimester of pregnancy is the largest, but the results are less clearly interpreted, with a negative coefficient for trimester two and none individually significant.

A similar analysis of the effect of conflict exposure on the probability of still birth (Table 4) shows that exposure to conflict *in utero* leads to a small decrease in the probability of still birth in absolute terms (0.15 ppts or just under 10 percent of the mean when controlling for maternal unobserved heterogeneity). Results in Columns (5) and (6) indicate that the decrease in the probability of still birth is driven by exposure to conflict up to mid-gestation, with a sign reversal for third trimester exposure, which is consistent with the interpretation that the probability of still birth decreases due to an increased probability of miscarriage. Contrary to the findings for miscarriage, however, controlling for maternal fixed effects does not affect the results.

The magnitude of the within-district effect of conflict exposure on the probability of miscarriage and still birth doubles and, in the case of still birth, becomes significant when restricting the sample to mothers with at least two pregnancies in the five years before the survey. In the case of miscarriage, the effect doubles again and becomes statistically significant when controlling for unobserved maternal heterogeneity. These findings suggest that the pregnancy outcomes of mothers who have comparatively close pregnancies are more affected by conflict, e.g., due to maternal depletion and more difficulties meeting the family's food and health care needs.

Combining the effect of conflict exposure *in utero* on both pregnancy outcomes (miscarriage and still birth) suggests "positive" selection on fetal health with a net decrease in the probability of live birth of about 0.7 ppts ($\approx 0.82 - 0.15$).

Further, indirect, evidence of the impact of exposure to conflict *in utero* on fetal loss and child health can be obtained by investigating the impact of conflict exposure on the gender of born children. It is well-established that the human male is more fragile than the female (Kraemer 2000). For this reason, and as hypothesized by Trivers and Willard (Trivers & Willard 1973), a poor health environment around and during pregnancy is expected to decrease the male-to-female sex ratio at birth. Evidence of a sex ratio skewed in favor of girls resulting from adverse maternal health shocks has been found in a range of circumstances (Fukuda et al. (1998); Almond & Mazumder (2008); Almond et al. (2007)). Results reported in Table 5 confirm that, when maternal unobserved heterogeneity is differenced-out,[10] the probability of a female live birth increases with exposure to conflict *in utero*, and the effect is larger for exposure to conflict up to month 5, consistent with my previous findings regarding miscarriages.

4.2 Effect of Conflict on Health Outcomes

In Table 6, I investigate the effect of exposure to conflict on the health of new born babies, as captured by size at birth and neonatal mortality, following the same approach as in Section 4.1.2. Overall exposure to conflict whilst in the womb does not appear

[10]For a discussion about the potential correlation between maternal characteristics and child gender, see Rosenfeld & Roberts (2004) and Fukuda et al. (2011).

to have a robust, statistically significant effect on either outcomes. However, children exposed to more intense fighting whilst in the womb tend to enjoy *better* health at and soon after birth. Consistent with the earlier findings regarding miscarriage, children exposed to higher conflict intensity up to mid-gestation are significantly less likely to be small at birth (by 0.9 ppts or 4.1 percent of the mean). On the contrary, there is a positive correlation between pre-conception, cumulated violence and neonatal mortality corresponding to an increase of 1.8 ppts or 31 percent of the mean, but the causal nature of this very large effect is unclear since it may be capturing long-term trends correlated with conflict intensity rather than a genuine causal effect. When considering the effect of exposure to conflict during each trimester of pregnancy, exposure to conflict during the second trimester appears to have the largest "positive" selection effect on small size at birth (-1 ppt) and neonatal mortality (-0.22 ppt). If taken at face value, these results could be interpreted as suggesting that babies who did not survive to birth due to a shock experienced in mid-gestation were those who would have been at highest risk of adverse health outcomes around birth. Caution should prevail, however, as splitting the gestation period in three trimesters also increases measurement error.

Similar to the previous results on pregnancy outcomes, restricting the sample to children with at least one sibling in the sample (i.e., comparing Columns (2) and (3)) leads to a sharp increase in the magnitude of the estimated effect of exposure to conflict, which gives further support to the hypothesis that the negative coefficients on conflict intensity *in utero* are due to selection on fetal health.

Before investigating the effect of exposure to conflict on child nutritional status using similar specifications as those used in this paper so far, I estimate a "standard" model formulation akin to those used, for instance, by Akresh et al. (2009) and Akresh et al. (2010) in some specifications, and where the treatment variable is the number of months of conflict since the birth of the index child, controlling for region and year of birth fixed effects. There is a strong correlation between the number of months of conflict and worse height-for-age, with a decrease of 0.02 standard deviation for each additional month of exposure (Table 7). These figures are surprisingly similar to those reported for identical specifications in Akresh et al. (2009) for Burundi (-0.035, Column (3) in their Table 5) and Akresh et al. (2010) for Ethiopia (-0.023, in Column (6) in their Table 2). The findings reported in Akresh et al. (2009) and Akresh et al. (2010) hold across a range of alternative robustness checks provided in these papers. However, when including a control for age in months (Column (2)), the effect of the number of months exposed to conflict disappears in my data. As explained in Section 3.2, estimates using the baseline specification of Equation 1 may be misleading because older children are more likely to have low height-for-age due to its cumulative nature.

From Column (3) onwards, conflict exposure variables are based on casualty counts. Monthly average conflict intensity since birth tends to have a negative effect on height for age, but this effect is only robust to controlling for age in months in the maternal fixed effects specifications. In this specification, maternal exposure to conflict before conception is negatively and significantly correlated with lower height for age (by 0.39 standard deviations of the reference population for an increase in conflict intensity akin to moving between the first and third terciles of the conflict-intensity district distribution). Exposure to conflict during the child's life leads to a HAZ decrease of 0.05 standard deviations of the reference population for a move between the first and third terciles of the conflict-intensity district distribution. The effect of exposure to conflict *in utero* is not statistically significant, and very small in magnitude (-0.02 s.d. for a move between

the first and third conflict terciles).

Similar to findings for pregnancy outcomes, the adverse effect of conflict on child nutritional status is larger in magnitude for children with at least one other sibling in the sample (i.e., in Column (7) compared to Column (6)), suggesting that families with more young children were more severely affected by the conflict.

When considering exposure to conflict at different periods of gestation, there is evidence of scarring from mid-gestation onwards, but similar to size at birth and neonatal mortality, higher conflict intensity in the second trimester of pregnancy is associated with better child nutritional outcomes, which would suggest that those pregnancies miscarried well into the gestational period would have been at highest risk of poor health outcomes.

Except for the effect of maternal exposure to conflict before conception, which is insignificant in all specifications, similar findings are obtained when replacing the height-for-age z-score with a binary indicator for having a HAZ below two standard deviations of the reference population median (namely, for a move between mean conflict exposure in the low conflict tercile to the high conflict tercile, a 2.7 ppts increase/0.82 ppts decrease/just under 1 ppt increase in the probability of being stunted for, respectively, exposure during the child's life/in the second trimester of pregnancy/in the third trimester of pregnancy).[11]

Across all health outcomes, the message from these results is that selection dominates for exposure in the second trimester, whilst scarring dominates in the third trimester, and the total effect of the two is undistinguishable from zero in the first trimester. This conclusion is consistent with the finding that exposure to conflict has tended to lead to more miscarriages up to mid-gestation, and suggests that those pregnancies miscarried well into the gestational period would have been at highest risk of poor health outcomes.

5 Robustness Checks

I first investigate whether there is any evidence of pre-conflict differential trends in fetal and child health by estimating the following equation on the sample of children conceived in the ten years preceding the start of the conflict:

$$Y_{idt} = \gamma_0 + \sum_{j=1}^{5} \gamma_j PreYear_{j,t} \times totaldeaths_d + X'_{idt}\gamma_X + PreYear'_t\gamma_y + D_d + v_{idt} \quad (2)$$

where Y_{idt} is an indicator of fetal or child health available for the entire fertility history of interviewed women (miscarriage, still birth, neonatal and infant mortality), $PreYear_t$ is a set of conception year dummies, $PreYear_{j,t}$ is the year dummy corresponding to the jth pre-conflict year, and $totaldeaths_d$ is the total number of casualties (for 1000 inhabitants) over the course of the entire conflict. If the γ_j are jointly insignificant, and, *a fortiori*, if each γ_j is not statistically different from zero, then we can conclude that there was no pre-conflict trend in the dependent variable systematically correlated with future conflict intensity. Results confirm that all γ_j are statistically insignificant individually as well as being jointly insignificant (the lowest p-value for the joint test is equal to 0.43). Similar tests were performed using the maternal fixed effects estimator, with similar conclusions.[12]

I then check whether findings for the preferred mother fixed effects specifications are

[11] Results are not reported for conciseness, but are available from the author upon request.

[12] Results are not reported for conciseness, but are available from the author upon request.

robust to a number of alterations, starting with excluding all controls except year fixed effects (and implicit maternal fixed effects). As can be seen in Panel A of Table 8, my previous results are robust to the exclusion of covariates.

I also try including a placebo treatment variable equal to the average monthly number of casualties during the same calendar period as the gestation period, but 12 months earlier. The magnitude and significance level of the coefficients on the conflict variables included in Equation 1 are largely unchanged, and the placebo effects are all insignificant except for small size at birth, where it indicates a correlation between conflict intensity 12 months before gestation and larger size at birth, so that caution should prevail when interpreting the effect of exposure to conflict *in utero* on size at birth as causal (Table 8, Panel B).

Finally, I control for district-specific linear trends, which is bound to reduce the precision with which the effect of conflict exposure is estimated, but should also provide results that are less likely due to omitted, time-varying confounding factors (Table 8, Panel C). Conclusions are unchanged, but three remarks are warranted. First, the marginally significant finding that exposure to conflict *in utero* reduces the probability of still birth becomes insignificant. Second, the correlation between neonatal mortality and maternal exposure to conflict before conception becomes insignificant. Finally, there is a very large increase in the magnitude of the effect of conflict before, during and after pregnancy on height-for-age, but not on the stunting indicator, suggesting the possibility of omitted variables correlated with heightened conflict and nutritional improvements for the non-poor only. This is consistent with the finding that inequality explains part of the variation in conflict intensity across districts of Nepal in Macours (2011).

For limited dependent variables, Equation 1 corresponds to the linear probability model, which does not take into account the nonlinear nature of the dependent variable, but provides more interpretable estimates. It is possible to estimate a nonlinear panel data model with (district or mother) fixed-effects that yields a consistent estimate of the effect of exposure to conflict on the log-odds ratio. However, this (conditional logit) model does not offer estimates for the fixed effects, and so it is not possible to estimate the partial effect of interest without making arbitrary assumptions on the value of the fixed effects (Wooldridge 2002). For rare events (neonatal mortality, miscarriage and still birth), I also estimated conditional logit models, which confirm the sign and significance of the findings obtained with the linear fixed-effects estimator.[13]

Finally, my conclusions are not altered when I include all children in the sample irrespective of whether or not their mothers already lived in their current place of residence when they became pregnant. This bolsters confidence in the robustness of my findings to conflict-induced migration.[14] Whilst increasing measurement error, including these children addresses the potential issue that some women may have migrated within the district due to conflict. It does not remedy the potential concern that mothers who migrated away from their district in response to violent conflict because we do not know where women have migrated from and therefore I have to assign conflict exposure on the basis of their district of residence at the time of the survey. Recall however that concerns over conflict-induced migration are less relevant in the case of Nepal than for many other conflict episodes because population displacement was comparatively rare (less than one percent of the population).

[13]Results are not reported for conciseness, but are available from the author upon request.

[14]Results are not reported for conciseness, but are available from the author upon request.

6 Analysis of Transmission Channels

6.1 Effect of Conflict on Health Care

Security concerns may discourage parents from seeking health care, and can disrupt the functioning of health facilities. In this section, I shed light on the impact of conflict intensity on use of key aspects of antenatal care (antenatal care checkups, iron and folic tablets, and anti-tetanus vaccination), delivery assistance, and child immunization.

Antenatal care data was only collected for the latest pregnancy of the respondent (if it occurred within 5 years of the survey). Therefore, it is not possible to compare siblings, so that estimates comprise a behavioral response as well as a compositional effect due to potential changes in the characteristics of mothers giving birth at times of conflict (Table A-1). For comparability with previous findings, I report both estimates for the whole sample (Columns (1), (3), and (5)) and findings for the (last child born amongst the) siblings sample (Columns (2), (4), and (6)). Overall, conflict intensity during pregnancy does not affect the number of antenatal care visits, nor the likelihood of receiving iron and folic acid tablets or a tetanus toxoid injection. Conflict intensity during the second trimester of pregnancy for children in the siblings sample has a very small, statistically significant negative effect on the number of antenatal care visits (-0.043 visits when going from the first to the third terciles of the conflict-intensity district distribution). Exposure to conflict before pregnancy, which could act as a deterrent to seek or give medical care, but as explained earlier is less likely to capture a causal effect, does not appear to have an effect either, except for its surprisingly positive correlation with the probability of receiving a tetanus toxoid injection (+3 to +5.4 ppts depending on specification).

Data on circumstances of delivery (place and attendant), as well as immunization data, are available for all children born within five years of the survey. For these variables I therefore also estimate maternal fixed effects models. Once again, conflict intensity does not appear to have had a negative effect on health seeking behavior. As seen in Table A-2 and Table A-3, estimated effects are small in magnitude, and almost always statistically insignificant. Exceptions are, for a move from the first to the third tercile of district conflict intensity: conflict intensity in the first trimester of pregnancy is associated with a 0.5 ppt decrease in the likelihood of giving birth without any assistance (within mother), which could be due to an increase in pregnancy-related problems which in some cases we have seen have led to miscarriages; conflict intensity in the third trimester of pregnancy leads to a 0.3 ppt increase in the likelihood of giving birth without assistance (within mother), which could be due to conflict very near the time of birth discouraging travel of mothers, their family, or birth attendants; children exposed to more conflict *in utero* are somewhat less likely to receive no immunization at all (0.2 ppt from tercile 1 to tercile 3); and children whose mothers have experienced more conflict before becoming pregnant are 1.3 ppt less likely to receive no immunization at all (from tercile 1 to tercile 3).

The overall lack of adverse effect of violent conflict on health care might seem surprising. However, when respondents were directly asked in the 2006 DHS why they did not deliver in a health facility, security reasons where blamed in only 1 percent of cases.

6.2 Effect of Conflict on Short-Term Nutritional Status

Here I consider the impact of cumulated conflict before the last three months before the survey separately from that of conflict in the last three months. Given the finding that exposure to conflict at different times of the pregnancy has a contemporaneous impact

on miscarriage, I am particularly interested in the effect of conflict on maternal nutrition in the short run. The identification strategy here differs from the main analysis because there is no sizeable variation in the short-term exposure to conflict before the survey within district for a given survey round. The estimation therefore relies on the variation within-district between DHS surveys. Conflict intensity in the three months preceding the survey is not correlated with short-term nutritional status of children (weight-for-height) and women (BMI). As shown in Table A-4, there is a positive and statistically significant correlation between conflict exposure up to three months before the survey and the probability of a child being wasted (1.95 ppt), but any estimated effect of recent conflict is negligible in magnitude (e.g., 0.19 ppt on the probability of being wasted, Column (2)) and insignificant.

6.3 Effect of Conflict on Female Labor Activity

Finally, Table A-5 shows that conflict intensity up to three months before the interview is correlated with an increased probability for mothers to be working in the seven days leading to the interview by 3.5 ppts from tercile 1 to tercile 3 (Column (1)), as also found in Menon & Rodgers (2011). Conflict intensity in the three months preceding the survey is however correlated with a lower probability of working (by 0.45 ppts). The latter effect is only marginally statistically significant. When restricting the sample to women who report having worked at some point during the last 12 months (Column (3)), I find that women are significantly more likely to be off work in the previous week where there is more conflict in the three months preceding the interview, albeit only by 0.7 ppt (for a move from tercile 1 to tercile 3 of district conflict intensity). Finally, conflict intensity does not appear to have any effect on the probability that mothers work from home.

7 Conclusion

This paper provides new evidence of the impact of exposure to civil conflict on child nutritional status, and extends the analysis of the impact of conflict on a range of health inputs and outcomes, from conception to age five, and paying particular attention to selection processes and to differentiating precisely between the effect of exposure of the child whilst *in utero* and exposure after birth.

I find direct evidence of selection away from fertility at times of more intense conflict by groups who were more directly targeted by the Maoists, namely dominant Brahmin and Chhetri castes and the (few) very highly educated, who have better health outcomes on average. However, a comparison of results holding constant unobserved district-level characteristics with estimates holding constant unobserved maternal-level characteristics shows that the adverse effect of conflict is more marked when differencing-out maternal time-invariant heterogeneity, thus suggesting that a stronger source of selection on maternal characteristics is at play, and one such that healthier women are more likely to become pregnant and give birth at times of violent conflict. In addition, I find that the adverse health effect of violent conflict is compounded by having more than one child within the relatively short period of time of five years.

I find direct evidence of an increased probability of miscarriage due to exposure to conflict in early to mid-pregnancy. More specifically, I estimate that going from the mean conflict exposure in the third of districts with lowest conflict intensity to that prevailing in the third of districts with highest conflict intensity increases the probability

of miscarriage by 0.82 ppts (12.9 percent of the mean). To cast this figure in a different light, it corresponds roughly to the increase in the probability of miscarriage when going from the 25th percentile of conflict intensity during pregnancy to the 75th percentile amongst children exposed to at least some conflict *in utero*. On the contrary, there is a small decrease in the probability of still birth (0.15 ppts), which is likely due to more selection via miscarriage. Although the possibility of increased fetal loss during times of civil conflict and war has been suggested in previous work (e.g., Wynn & Wynn (1993); Rajab et al. (2000); Roseboom et al. (2001)), to the best of my knowledge this is the first study showing direct, arguably causal, evidence of this phenomenon.

Exposure to conflict in the first few years of life has an adverse effect on child nutritional status when holding unobserved maternal characteristics constant, at least for the sample of children with a sibling born within five years, who are a large, but more vulnerable, subsample. However, exposure to conflict *in utero* has both scarring and selection effects. As conflict intensity increases, the likelihood of miscarriage increases, and so a smaller share of the frailer fetuses is carried to term. This selection effect tends to dominate in the second trimester of pregnancy, whilst scarring effects are stronger in the third trimester.

For all outcomes, the adverse health effect of violent conflict is compounded by having more than one child within the relatively short period of time of five years. Even so, and after controlling for maternal fixed effects, the effects estimated are comparatively small (e.g., a 2.7 ppts increase in the stunting rate for a mean of 57 percent, when going from the low-conflict district group to the high-conflict district group). The results reported here however provide suggestive evidence of a potentially large effect of maternal exposure to conflict before conception on neonatal mortality, although the causal nature of the observed correlation is unclear.

Interestingly, use of antenatal care, (medical) help with delivery, and immunization do not appear to decrease when conflict intensifies. In addition, there is no evidence of acute nutritional distress caused by conflict in the three months preceding the survey, whereas there is suggestive evidence of reduced female labor force participation where conflict was recently more intense.

Taken together, these results show that selection on parental characteristics and fetal health are non-negligible factors for prolonged conflicts, even in the context of a moderate intensity of violence. Furthermore, they suggest that exposure to conflict intensity around and after birth has noticeable scarring effects on child nutritional status even in the context of conflicts of moderate intensity. On the contrary, results indicate that use of health facilities are not necessarily dramatically disrupted, possibly due in part to a reduced opportunity cost of maternal time. There is no evidence either of short-term malnutrition to support the hypothesis that maternal malnutrition mediates the effect of civil conflict at specific times during pregnancy. The main residual candidate mechanism which could explain the contemporaneous effect of conflict on miscarriage therefore is maternal stress.

Since conflict cannot be randomized, a difference-in-difference approach is generally used to obtain plausibly causal estimates, as it is the case here. This however comes with the caveat that civil conflict may have consequences for the whole country, so that within-district estimates are likely to underestimate the total effect of conflict. Further research trying to pinpoint the causal effect of cumulated conflict before conception would be welcome.

Nevertheless, this study suggests two important policy implications. First, in conflicts of moderate intensity, food aid may be more efficiently targeted if focussing on families

with several young children and pregnant women. Second, it stresses the need to consider public health policies aimed at supporting women in conflict situations to deal with the trauma of pregnancy loss.

Table 1: Summary Statistics

District Conflict-Intensity Tercile	(1) Low			(2) Intermediate			(3) High		
	mean	sd	count	mean	sd	count	mean	sd	count
Live births sample									
Explained variables									
=1 if female	0.508		3894	0.481		3404	0.498		3300
=1 if neonatal death	0.035		3894	0.035		3404	0.036		3300
=1 if small baby	0.166		3894	0.241		3404	0.239		3300
Delivered by doctor or nurse	0.183		3894	0.103		3404	0.085		3300
No delivery help	0.051		3894	0.121		3404	0.103		3300
Antenatal visits	2.355	2.5260	2771	1.799	2.0218	2355	1.486	1.8381	2333
Iron tablets during pregnancy	0.431		2775	0.358		2357	0.313		2334
Tetanus immunization during pregnancy	0.718		2774	0.543		2357	0.509		2334
Height-for-age z-score	-1.997	1.3784	3548	-2.197	1.3332	3109	-2.204	1.2638	3016
Weight-for-height z-score	-0.911	1.0540	3548	-0.733	1.0693	3109	-0.766	1.0427	3016
=1 if stunted (HAZ<-2 s.d.)	0.513		3548	0.577		3109	0.582		3016
=1 if wasted (WHZ<-2 s.d.)	0.139		3548	0.109		3109	0.102		3016
No immunization	0.011		2917	0.014		2541	0.028		2514
Some immunization	0.231		2917	0.230		2541	0.247		2514
Full immunization	0.757		2917	0.756		2541	0.726		2514
Selected Pregnancy characteristics									
=1 if first pregnancy	0.225		3894	0.210		3404	0.213		3300
=1 if second pregnancy	0.229		3894	0.216		3404	0.226		3300
=1 if third pregnancy	0.193		3894	0.169		3404	0.164		3300
=1 if fourth pregnancy	0.126		3894	0.127		3404	0.126		3300
=1 if fifth pregnancy and higher	0.226		3894	0.278		3404	0.271		3300
Maternal age at conception	24.107	5.9500	3894	24.886	6.3952	3404	24.485	6.3171	3300
Selected Maternal characteristics									
=1 if urban	0.186		3894	0.097		3404	0.118		3300
=1 if no education	0.663		3894	0.716		3404	0.755		3300
=1 if primary education	0.149		3894	0.154		3404	0.140		3300
=1 if secondary education	0.165		3894	0.116		3404	0.098		3300
=1 if higher education	0.023		3894	0.014		3404	0.007		3300

(Continued on next page)

(Continued)

District Conflict-Intensity Tercile	(1) Low			(2) Intermediate			(3) High		
	mean	sd	count	mean	sd	count	mean	sd	count
=1 if Dalit	0.170	0.3757	3894	0.140	0.3472	3404	0.165	0.3716	3300
=1 if Brahmin or Chhetri	0.213		3894	0.380		3404	0.422		3300
=1 if Tarai/Madhesi	0.256		3894	0.046		3404	0.013		3300
=1 if Newar	0.034		3894	0.032		3404	0.037		3300
=1 if Janajati	0.194		3894	0.395		3404	0.334		3300
=1 if Muslim	0.107		3894	0.005		3404	0.024		3300
=1 if Other caste	0.025		3894	0.001		3404	0.005		3300
Wealth quintile									
=1 if first quintile(lowest)	0.154		3894	0.366		3404	0.360		3300
=1 if second quintile	0.203		3894	0.191		3404	0.262		3300
=1 if third quintile	0.229		3894	0.157		3404	0.164		3300
=1 if fourth quintile	0.206		3894	0.192		3404	0.152		3300
=1 if fifth quintile	0.207		3894	0.094		3404	0.062		3300
Conflict exposure - district casualties per 1000 inhabitants									
Before pregnancy, total	0.039	0.0783	3894	0.118	0.2124	3404	0.424	0.7518	3300
During pregnancy, monthly average	0.002	0.0033	3894	0.005	0.0086	3404	0.011	0.0207	3300
Trimester 1, monthly average	0.002	0.0041	3894	0.005	0.0113	3404	0.011	0.0318	3300
Trimester 2, monthly average	0.002	0.0041	3894	0.005	0.0113	3404	0.011	0.0301	3300
Trimester 3, monthly average	0.002	0.0041	3894	0.005	0.0116	3404	0.011	0.0262	3300
Conception to conception+4, monthly average	0.002	0.0039	3894	0.005	0.0106	3404	0.011	0.0287	3300
Conception+5 to conception+9, monthly average	0.002	0.0039	3894	0.005	0.0109	3404	0.011	0.0248	3300
Since birth, monthly average	0.002	0.0027	3894	0.004	0.0048	3404	0.008	0.0107	3300
Pregnancy sample									
Explained variables									
=1 if Miscarriage	0.054		4395	0.047		3860	0.053		3773
=1 if Still birth	0.012		4209	0.009		3709	0.014		3624
Duration of pregnancy if miscarried									
1 Month	0.076		237	0.011		183	0.025		199
2 Months	0.211		237	0.164		183	0.156		199
3 Months	0.325		237	0.437		183	0.302		199

(Continued on next page)

(Continued)

District Conflict-Intensity Tercile	(1) Low			(2) Intermediate			(3) High		
	mean	sd	count	mean	sd	count	mean	sd	count
4 Months	0.190		237	0.175		183	0.256		199
5 Months	0.110		237	0.115		183	0.141		199
6 Months	0.059		237	0.093		183	0.075		199
7 Months	0.008		237	0.000		183	0.035		199
8 Months	0.013		237	0.005		183	0.010		199
9 Months	0.008		237	0.000		183	0.000		199

Source: Author's calculations using Nepal DHS 2001 and DHS 2006. Antenatal care data were only collected for the last live born child if birth occurred within five years of the survey. Immunization data were only collected for children alive at the time of the survey, and the immunization sample is restricted here to children aged at least 1 year old. Following WHO guidelines, children are considered fully immunized when they have received one dose of the tuberculosis vaccine (BCG) and measles vaccine, and three doses of the polio and DPT vaccines. The Nepal DHS contains 96 ethnicity categories. Here they are grouped following Bennett, L. and Ram Dahal, D. and Govindasamy, P. (2008). Binary indicators for wealth quintiles are provided in the DHS based on a principal component analysis of (i) ownership of consumer items such as television, bicycle, car, (ii) dwelling characteristics including source of drinking water, sanitation and type of housing materials. The miscarriage and still birth indicators are equal to zero for live births, and set to missing if the mother does not answer "No" when asked if any action was taken to end the pregnancy.

Table 2: Selection on Parental Characteristics

	(1)	(2)	(3)	(4)	(5)
Wealth Quintile	First (Lowest)	Second	Third	Fourth	Fifth
Before conception	0.0591	-0.0402	0.0099	-0.0256	-0.0032
(total)	(0.0399)	(0.0336)	(0.0266)	(0.0245)	(0.0147)
During pregnancy	-0.3579	0.2936	0.2284	0.0730	-0.2371
	(0.3700)	(0.3647)	(0.3484)	(0.3300)	(0.1898)
Mean Y_{idt}	0.286	0.216	0.184	0.185	0.128

	(1)	(2)	(3)	(4)	(5)
Explained Variable	=1 if Mother has No Education	=1 if Mother has 1ary Education	=1 if Mother has 2ary Education	=1 if Mother has Higher Education	=1 if Urban
Before conception	0.0283	0.0023	-0.0165	-0.0142**	0.0001
(total)	(0.0216)	(0.0150)	(0.0177)	(0.0062)	(0.0317)
During pregnancy	0.0587	0.2106	-0.1404	-0.1289*	-0.0368
	(0.2075)	(0.1765)	(0.1742)	(0.0768)	(0.2877)
Mean Y_{idt}	0.708	0.148	0.129	0.016	0.139

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Caste/Ethnicity	=1 if Brahmin/	=1 if Madhesi	=1 if Dalit	=1 if Newar	=1 if Janajati	=1 if Muslim	=1 if Other
Caste/Ethnicity	Chhetri						Caste
Before conception	-0.0903**	0.0238*	0.0091	0.0050	0.0455	0.0109	-0.0040
(total)	(0.0390)	(0.0142)	(0.0367)	(0.0082)	(0.0305)	(0.0197)	(0.0057)
During pregnancy	-0.7780**	0.2494	0.0960	0.2109	0.2059	0.0657	-0.0498
	(0.3482)	(0.1582)	(0.4090)	(0.1391)	(0.3139)	(0.2102)	(0.0566)
Mean Y_{idt}	0.342	0.110	0.161	0.034	0.296	0.047	0.011
Observations	12490	12490	12490	12490	12490	12490	12490

All regressions are estimated using the linear (district) fixed-effect estimator and include dummies for year of conception, month of conception and controls for pregnancy characteristics X_{idt}, namely age of mother at conception and its square, pregnancy order indicators (for second, third, fourth, and "five and above"), and 11 calendar month of conception dummies. District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. * p<0.10, ** p<0.05, *** p<0.01

Table 3: Effect of Exposure to Violent Conflict on the Probability of Miscarriage

	=1 if Miscarriage					
	(1)	(2)	(3)	(4)	(5)	(6)
Before conception	0.0009	0.0017	0.0055	0.0258	0.0203	0.0214
(total)	(0.0079)	(0.0078)	(0.0121)	(0.0197)	(0.0214)	(0.0206)
During pregnancy	0.2243	0.2361	0.4555	0.9150***		
	(0.2684)	(0.2679)	(0.3289)	(0.3049)		
Conception to					0.6806**	
conception+4					(0.2747)	
Conception+5					0.0714	
to conception+9					(0.4117)	
First trimester						0.5547
						(0.3558)
Second trimester						-0.2213
						(0.3117)
Third trimester						0.4355
						(0.3339)
Panel variable	District	District	District	Mother	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Maternal characteristics	No	Yes	Yes	No	No	No
Observations	12028	12028	7268	7268	7268	7268
Number of Groups	75	75	74	3324	3324	3324
No. of clusters	75	75	74	74	74	74
R-squared	0.0063	0.0078	0.0111	0.0446	0.0444	0.0444
Mean Y_{idt}	0.051	0.051	0.064	0.064	0.064	0.064

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: a binary indicator for urban households, maternal education dummies (primary, secondary, higher), and caste/ethnicity indicators (Brahmin/Chhetri, Other Tarai/Madhesi, Newar, Janajati, Muslim, other castes). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. * p<0.10, ** p<0.05, *** p<0.01

Table 4: Effect of Exposure to Violent Conflict on the Probability of Stillbirth

	=1 if Stillbirth					
	(1)	(2)	(3)	(4)	(5)	(6)
Before conception	-0.0061	-0.0061	-0.0107	-0.0025	-0.0017	-0.0014
(total)	(0.0056)	(0.0057)	(0.0065)	(0.0052)	(0.0052)	(0.0048)
During pregnancy	-0.0906	-0.0912	-0.1835**	-0.1642*		
	(0.0790)	(0.0812)	(0.0911)	(0.0874)		
Conception					-0.1757*	
to conception+4					(0.0890)	
Conception+5					0.0267	
to conception+9					(0.0446)	
First trimester						-0.1083
						(0.0725)
Second trimester						-0.0873**
						(0.0352)
Third trimester						0.0658
						(0.0448)
Panel variable	District	District	District	Mother	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Maternal characteristics	No	Yes	Yes	No	No	No
Observations	11542	11542	6715	6715	6715	6715
Number of Groups	75	75	74	3126	3126	3126
No. of clusters	75	75	74	74	74	74
R-squared	0.0076	0.0082	0.0131	0.0114	0.0116	0.0116
Mean Y_{idt}	0.012	0.012	0.015	0.015	0.015	0.015

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. * p<0.10, ** p<0.05, *** p<0.01

Table 5: Effect of Exposure to Violent Conflict on the Probability of a Female Birth

	=1 if Female					
	(1)	(2)	(3)	(4)	(5)	(6)
Before conception	-0.0058	-0.0056	-0.0068	0.0475	0.0467	0.0335
(total)	(0.0182)	(0.0177)	(0.0208)	(0.0508)	(0.0512)	(0.0479)
During pregnancy	-0.2894	-0.3195	-0.2260	1.7782*		
	(0.5057)	(0.5010)	(0.6129)	(0.9869)		
Conception					1.1039**	
to conception+4					(0.5281)	
Conception+5					0.6715	
to conception+9					(0.7757)	
First trimester						0.8685
						(0.5764)
Second trimester						-0.3960
						(0.3279)
Third trimester						0.9600
						(0.5939)
Panel variable	District	District	District	Mother	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Maternal characteristics	No	Yes	Yes	No	No	No
Observations	10598	10598	5630	5630	5630	5630
Number of Groups	75	75	74	2679	2679	2679
No. of clusters	75	75	74	74	74	74
R-squared	0.0031	0.0047	0.0064	0.0100	0.0101	0.0101
Mean Y_{idt}	0.497	0.497	0.517	0.517	0.517	0.517

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. The DHS only collected child gender data for live births. * p<0.10, ** p<0.05, *** p<0.01

Table 6: Effect of Exposure to Violent Conflict on Newborn Health

	=1 if Small at Birth					
	(1)	(2)	(3)	(4)	(5)	(6)
Before conception	-0.0140	-0.0157	-0.0220	-0.0191	-0.0180	-0.0114
(total)	(0.0253)	(0.0250)	(0.0311)	(0.0304)	(0.0304)	(0.0290)
During pregnancy	-0.2267	-0.2088	-0.7773	-1.4501		
	(0.5486)	(0.5536)	(0.5565)	(0.8806)		
Conception					-1.0007***	
to conception+4					(0.3629)	
Conception+5					-0.4458	
to conception+9					(0.7912)	
First trimester						-0.4674
						(0.4032)
Second trimester						-1.1656**
						(0.4639)
Third trimester						0.5725
						(0.9024)
R-squared	0.0089	0.0146	0.0232	0.0216	0.0218	0.0231
Mean Y_{idt}	0.213	0.213	0.219	0.219	0.219	0.219
	=1 if Neonatal Death					
	(1)	(2)	(3)	(4)	(5)	(6)
Before conception	0.0083	0.0075	0.0165	0.0471**	0.0472**	0.0423**
(total)	(0.0094)	(0.0094)	(0.0144)	(0.0192)	(0.0191)	(0.0201)
During pregnancy	-0.1482	-0.1479	-0.3488*	-0.2833		
	(0.1344)	(0.1345)	(0.1891)	(0.4432)		
Conception					-0.1645	
to conception+4					(0.2655)	
Conception+5					-0.1185	
to conception+9					(0.3648)	
First trimester						-0.0747
						(0.2807)
Second trimester						-0.2464*
						(0.1331)
Third trimester						-0.1211
						(0.3249)
R-squared	0.0081	0.0094	0.0200	0.0311	0.0311	0.0315
Mean Y_{idt}	0.035	0.035	0.050	0.050	0.050	0.050
Panel variable	District	District	District	Mother	Mother	Mother
Maternal characteristics	No	Yes	Yes	No	No	No
Observations	10598	10598	5630	5630	5630	5630
Number of Groups	75	75	74	2679	2679	2679

All regressions are estimated using the panel data fixed effects estimator and include controls of pregnancy characteristics (as listed under Table 2), year of conception fixed effects, calendar month of conception dummies, and a constant. Maternal Characteristics: as listed under Table 3. District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. * p<0.10, ** p<0.05, *** p<0.01

Table 7: Effect of Exposure to Violent Conflict on Child Height-for-Age Z-score (HAZ)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Months of conflict	-0.0203***	0.0024							
since birth	(0.0039)	(0.0049)							
Before conception			-0.0120	0.0075	-0.0452	-0.0192	-0.2447**	-0.2439**	-0.2304**
(total)			(0.0994)	(0.0969)	(0.1098)	(0.0863)	(0.1125)	(0.1123)	(0.1096)
During pregnancy			-0.7033	-0.7139	-1.3056	-0.8725	-2.3954		
			(1.3220)	(1.3970)	(2.1621)	(2.2561)	(2.0987)		
Since birth			-5.4457	-6.5050*	-12.2262***	-1.7395	-8.2337**	-7.9277**	-7.6893*
			(3.4502)	(3.3222)	(4.0446)	(3.5864)	(3.7982)	(3.8272)	(4.0912)
Conception								-0.2857	
to conception+4								(1.6091)	
Conception+5								-2.1320**	
to conception+9								(1.0019)	
First trimester									-1.4445
									(1.5793)
Second trimester									1.9166**
									(0.8838)
Third trimester									-3.3333***
									(1.0748)
R-squared	0.1302	0.1401	0.1408	0.1699	0.1760	0.1970	0.3222	0.3224	0.3237
Mean Y_{idt}	-2.126	-2.126	-2.126	-2.126	-2.181	-2.181	-2.181	-2.181	-2.181
Panel variable	District	District	District	District	District	District	Mother	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	No	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Age in month	No	Yes	No	No	No	Yes	Yes	Yes	Yes
Pregnancy characteristics	No	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Maternal characteristics	No	No	No	Yes	Yes	Yes	No	No	No
Observations	9673	9673	9673	9673	4577	4577	4577	4577	4577
Number of Groups	75	75	75	75	74	74	2211	2211	2211

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. * p<0.10, ** p<0.05, *** p<0.01

Table 8: Robustness Checks

	(1) Miscarriage	(2) Still Birth	(3) Female	(4) Neo. Death	(5) Small Baby	(6) HAZ	(7) HAZ<-2
Panel A: No Controls							
Before conception	0.0242	-0.0052	0.0395	0.0472**	-0.0133	-0.1612	0.0098
(total)	(0.0179)	(0.0043)	(0.0496)	(0.0187)	(0.0304)	(0.1087)	(0.0458)
During pregnancy	0.9148***	-0.1694**	1.7966*	-0.2587	-1.3186	-1.2255	-0.0954
	(0.2953)	(0.0827)	(0.9905)	(0.4199)	(0.8711)	(1.9736)	(0.7633)
Since birth						-7.2249	4.1896***
						(4.5210)	(1.1296)
Panel variable	Mother	Mother	Mother	Mother	Mother	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.0025	0.0033	0.0067	0.0104	0.0068	0.3093	0.2199
Panel B: Placebo experiments							
Before conception (total)	0.0295	-0.0011	0.0636	0.0460**	0.0120	-0.2452**	0.0478
	(0.0253)	(0.0049)	(0.0575)	(0.0206)	(0.0334)	(0.1169)	(0.0509)
During pregnancy	0.9324***	-0.1537*	1.8856*	-0.2905	-1.2429	-2.3984	0.3476
	(0.2997)	(0.0840)	(1.0262)	(0.4388)	(0.8522)	(2.1008)	(0.7746)
During pregnancy - 12 months	-0.1611	-0.0612	-0.7316	0.0497	-1.4110***	0.0252	-0.4150
	(0.6468)	(0.0480)	(0.8145)	(0.2617)	(0.4886)	(1.0622)	(0.6016)
Since birth						-8.2273**	4.4014***
						(3.7789)	(1.0739)
Age in months						-0.0450***	0.0159***
						(0.0072)	(0.0029)
Panel variable	Mother	Mother	Mother	Mother	Mother	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.0447	0.0114	0.0102	0.0311	0.0230	0.3222	0.2309
Panel C: Controlling for district-specific linear trend							
Before conception (total)	0.0237	-0.0056	0.0908	0.0353	-0.0681	-0.6709***	0.1133
	(0.0315)	(0.0118)	(0.0759)	(0.0270)	(0.0571)	(0.2400)	(0.0880)
During pregnancy	0.9655***	-0.1379	2.1937**	-0.5177	-1.5403	-5.4305**	0.7693
	(0.3443)	(0.0984)	(1.0361)	(0.4871)	(1.1595)	(2.6071)	(0.9895)
Since birth						-15.1355**	5.0661**

(Continued on next page)

(Continued)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
						(6.7435)	(1.9438)
Age in months						-0.0572***	0.0194***
						(0.0073)	(0.0031)
Panel variable	Mother	Mother	Mother	Mother	Mother	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes	Yes
District yearly trend	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.0572	0.0253	0.0280	0.0452	0.0445	0.3564	0.2616
Observations	7268	6715	5630	5630	5630	4577	4577
Number of Groups	3324	3126	2679	2679	2679	2211	2211
No. of clusters	74	74	74	74	74	74	74
Mean Y_{idt}	0.064	0.015	0.517	0.050	0.219	-2.181	0.573

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. * p<0.10, ** p<0.05, *** p<0.01

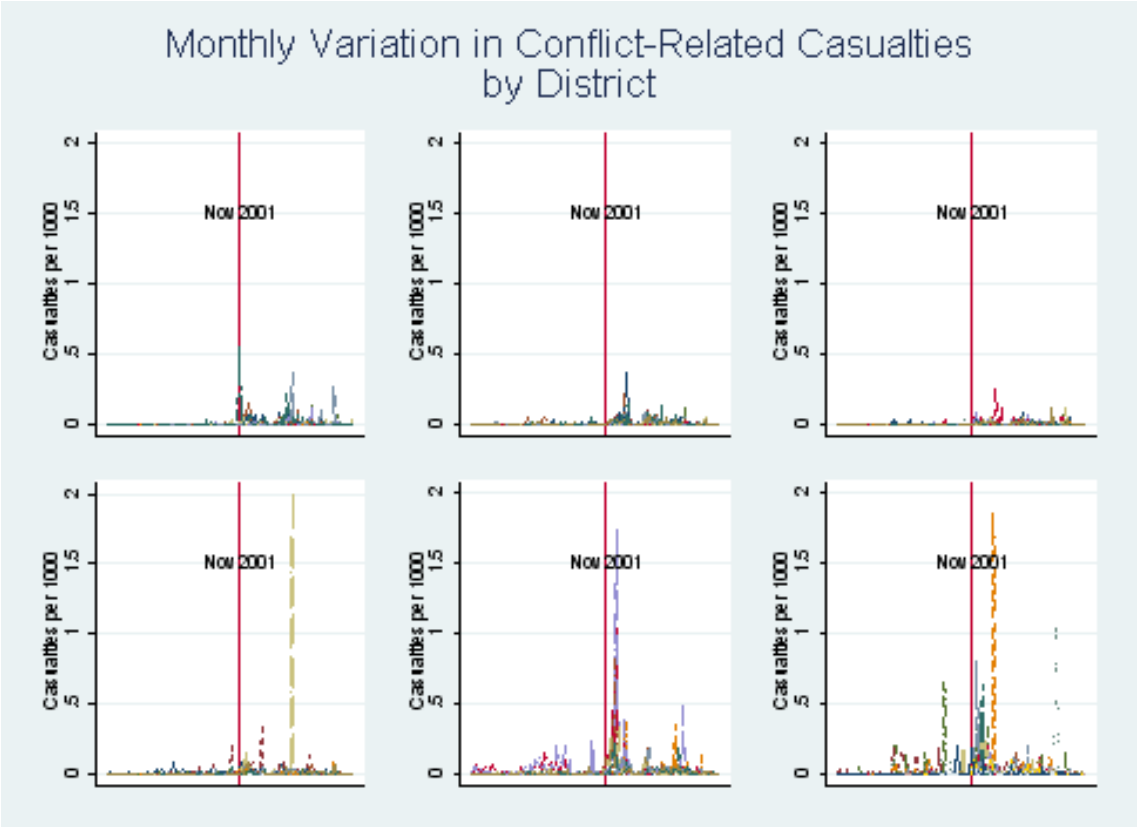

Figure 1: Within-District Variation in Casualties

The 75 Nepalese districts are split in 6 groups and represented in separate graphs.

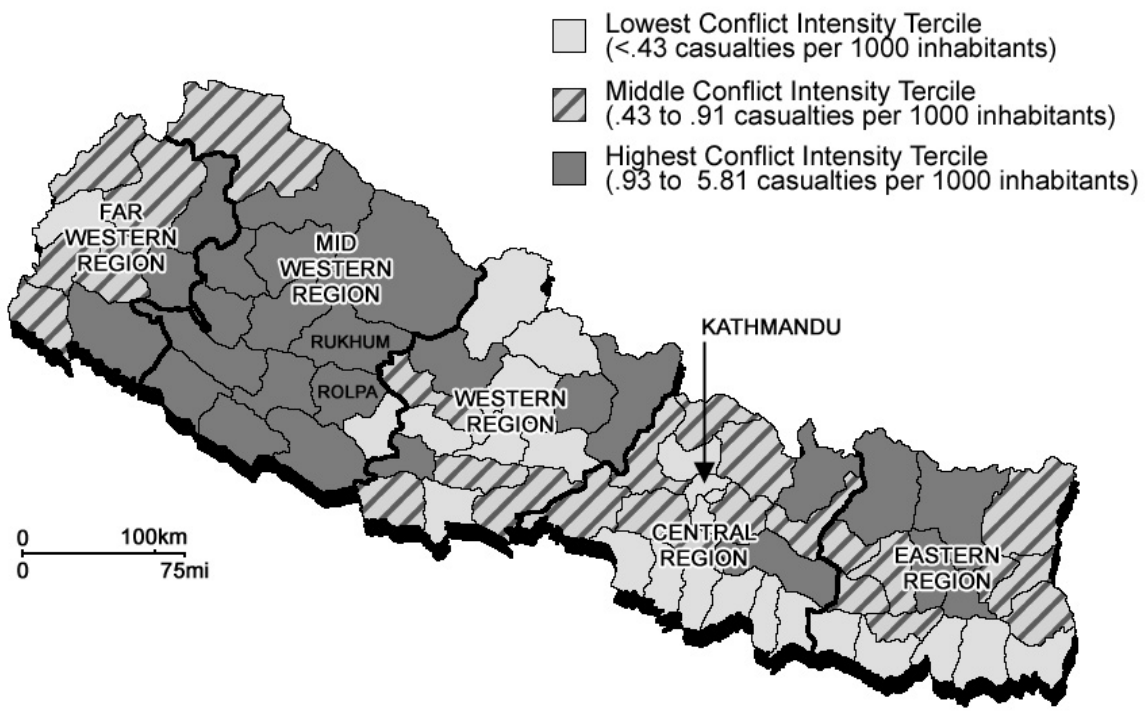

Figure 2: Between-District Variation in Casualties

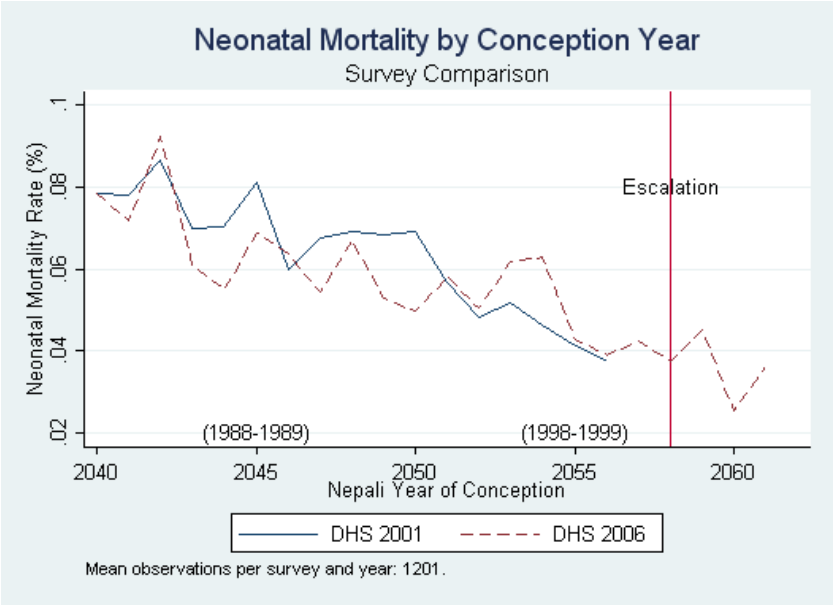

Figure 3: Reported Neonatal Mortality Rates by Year and Survey Round

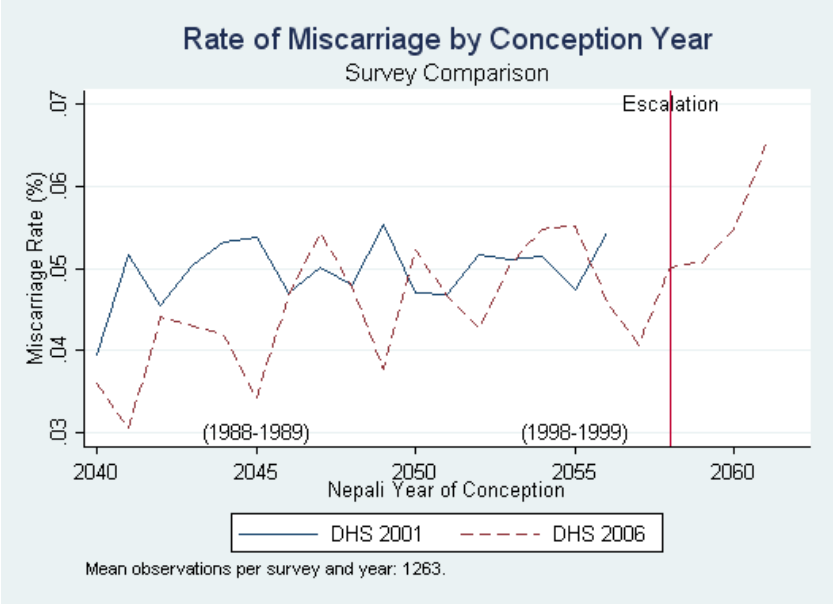

Figure 4: Reported Miscarriage Rates by Year and Survey Round

References

Agadjanian, V. & Prata, N. (2002), 'War, peace, and fertility in Angola', *Demography* **39**(2), 215–231.

Akbulut-Yuksel, M. (2009), 'Children of war: The long-run effects of large-scale physical destruction and warfare on children', *IZA Discussion Paper No. 4407* .

Akresh, R., Bundervoet, T. & Verwimp, P. (2009), 'Health and civil war in rural Burundi', *Journal of Human Resources* **44**(2), 536–63.

Akresh, R., Lucchetti, L. & Thirumurthy, H. (2010), 'Wars and Child Health: Evidence from the Eritrean-Ethiopian Conflict', *IZA Discussion Paper No. 5558* .

Alderman, H., Hoddinott, J. & Kinsey, B. (2006), 'Long term consequences of early childhood malnutrition', *Oxford Economic Papers* **58**(3), 450.

Allanson, B., Jennings, B., Jacques, A., Charles, A., Keil, A. & Dickinson, J. (2010), 'Infection and fetal loss in the mid-second trimester of pregnancy', *Australian and New Zealand Journal of Obstetrics and Gynaecology* **50**(3), 221–225.

Almond, D. (2006), 'Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 US population', *Journal of Political Economy* **114**(4), 672–712.

Almond, D. & Currie, J. (forthcoming), 'Killing me softly: The fetal origins hypothesis', *The Journal of Economic Perspectives* .

Almond, D., Edlund, L., Li, H. & Zhang, J. (2010), *Long-term effects of the 1959-1961 China famine: Mainland China and Hong Kong*, The Economic Consequences of Demographic Change in East Asia, Chicago: University of Chicago Press, pp. 321–345.

Almond, D., Edlund, L. & Palme, M. (2007), 'Chernobyl's subclinical legacy: Prenatal exposure to radiocative fallout and school outcomes in sweden', *NBER Working Paper No. 13347* .

Almond, D. & Mazumder, B. (2008), 'The effects of maternal fasting during ramadan on birth and adult outcomes', *Federal Reserve Bank of Chicago Working Paper WP-07-22* .

Ancel, P., Saurel-Cubizolles, M., Di Renzo, G., Papiernik, E. & Breart, G. (2000), 'Risk factors for 14-21 week abortions: a case-control study in europe.', *Human reproduction (Oxford, England)* **15**(11), 2426–2432.

Austin, M. & Leader, L. (2000), 'Maternal stress and obstetric and infant outcomes: epidemiological findings and neuroendocrine mechanisms', *Australian and New Zealand Journal of Obstetrics and Gynaecology* **40**(3), 331–337.

Barker, D. J. (1995), 'Fetal origins of coronary heart disease', *BMJ* **311**(6998), 171.

Beckett, M., Da Vanzo, J., Sastry, N., Panis, C. & Peterson, C. (2001), 'The quality of retrospective data: an examination of long-term recall in a developing country', *The Journal of Human Resources* **36**(3), 593–625.

Bennett, L. and Ram Dahal, D. and Govindasamy, P. (2008), *Caste, Ethnic and Regional Identity in Nepal: Further Analysis of the 2006 Nepal Demographic and Health Survey*, Calverton, Maryland, USA.

Beun, M. & Neupane, B. (2003), 'Impact of Conflict on Accessibility of EOC Health Services', *Nepal: DFID* .

Bhalotra, S. & van Soest, A. (2008), 'Birth-spacing, fertility and neonatal mortality in India: Dynamics, frailty, and fecundity', *Journal of Econometrics* **2**(143), 274–290.

Bispham, J., Gopalakrishnan, G., Dandrea, J., Wilson, V., Budge, H., Keisler, D., Broughton Pipkin, F., Stephenson, T. & Symonds, M. (2003), 'Maternal endocrine adaptation throughout pregnancy to nutritional manipulation: consequences for maternal plasma leptin and cortisol and the programming of fetal adipose tissue development', *Endocrinology* **144**(8), 3575.

Blattman, C. & Miguel, E. (2010), 'Civil war', *Journal of Economic Literature* **48**(1), 3–57.

Bohara, A., Mitchell, N. & Nepal, M. (2006), 'Opportunity, democracy, and the exchange of political violence', *Journal of conflict resolution* **50**(1), 108–128.

Cai, Y. & Feng, W. (2005), 'Famine, social disruption, and involuntary fetal loss: evidence from Chinese survey data', *Demography* **42**(2), 301–322.

Camacho, A. (2007), 'Stress and Birth Weight: Evidence from terrorist attacks in Colombia', *Universidad de los Andes Documento CEDE 2007-18* .

Cnattingius, S., Haglund, B. & Kramer, M. (1998), 'Differences in late fetal death rates in association with determinants of small for gestational age fetuses: population based cohort study', *Bmj* **316**(7143), 1483.

Collins, S. (2006), 'Assessing the health implications of Nepal's ceasefire', *Lancet* **368**, 907–908.

Dehejia, R. & Lleras-Muney, A. (2004), 'Booms, Busts, and Babies' Health', *Quarterly Journal of Economics* **119**(3), 1091–1130.

Devkota, M. D. (2005), 'Nepal Health Sector Program', *Nepal: World Bank Health Nutrition and Population* .

Do, Q. & Iyer, L. (forthcoming), 'Geography, Poverty and Conflict in Nepal', *Journal of Peace Research* .

El Metwalli, A., Badawy, A., El Baghdadi, L. & El Wehady, A. (2001), 'Occupational physical activity and pregnancy outcome', *European Journal of Obstetrics & Gynecology and Reproductive Biology* **100**(1), 41–45.

Eloundou-Enyegue, P., Stokes, C. & Cornwell, G. (2000), 'Are there crisis-led fertility declines? Evidence from central Cameroon', *Population Research and Policy Review* **19**(1), 47–72.

Figa-Talamanca, I. (1984), 'Spontaneous abortions among female industrial workers', *International archives of occupational and environmental health* **54**(2), 163–171.

Fukuda, M., Fukuda, K., Shimizu, T. & Møller, H. (1998), 'Decline in sex ratio at birth after kobe earthquake.', *Human reproduction* **13**(8), 2321.

Fukuda, M., Fukuda, K., Shimizu, T., Nobunaga, M., Grete Byskov, A. & Yding Andersen, C. (2011), 'The sex ratio of offspring is associated with the mothers age at menarche', *Human Reproduction* .

Garcia-Enguidanos, A., Calle, M., Valero, J., Luna, S. & Dominguez-Rojas, V. (2002), 'Risk factors in miscarriage: a review', *European Journal of Obstetrics & Gynecology and Reproductive Biology* **102**(2), 111–119.

Glewwe, P. & Miguel, E. (2008), 'The impact of child health and nutrition on education in less developed countries, in Handbook of Development Economics, vol. 4, edited by T. Paul Schultz and John Strauss eds'.

Glynn, L., Wadhwa, P., Dunkel-Schetter, C., Chicz-DeMet, A. & Sandman, C. (2001), 'When stress happens matters: effects of earthquake timing on stress responsivity in pregnancy', *American Journal of Obstetrics and Gynecology* **184**(4), 637–642.

Helgstrand, S. & Andersen, A.-M. N. (2005), 'Maternal underweight and the risk of spontaneous abortion', *Acta Obstetricia et Gynecologica Scandinavica* **84**(12), 1197–1201.

Herrmann, T. S., Siega-Riz, A. M., Hobel, C. J., Aurora, C. & Dunkel-Schetter, C. (2001), 'Prolonged periods without food intake during pregnancy increase risk for elevated maternal corticotropin-releasing hormone concentrations', *American Journal of Obstetrics and Gynecology* **185**(2), 403–412.

Hobel, C. & Culhane, J. (2003), 'Role of psychosocial and nutritional stress on poor pregnancy outcome', *The Journal of nutrition* **133**(5), 1709S.

Hobel, C., Dunkel-Schetter, C., Roesch, S., Castro, L. & Arora, C. (1999), 'Maternal plasma corticotropin-releasing hormone associated with stress at 20 weeks' gestation in pregnancies ending in preterm delivery', *American Journal of Obstetrics and Gynecology* **180**(1), S257–S263.

Human Rights Watch (2004), 'Between a rock and a hard place: Civilians struggle to survive in nepal's civil war', *http://www.hrw.org/en/reports/2004/10/06/between-rock-and-hard-place?print* . *accessed 10th Feb 2011* .

Incerpi, M., Miller, D., Samadi, R., Settlage, R. & Goodwin, T. (1998), 'Stillbirth evaluation: what tests are needed?', *American journal of obstetrics and gynecology* **178**(6), 1121–1125.

Jayaraman, A., Gebreselassie, T. & Chandrasekhar, S. (2009), 'Effect of Conflict on Age at Marriage and Age at First Birth in Rwanda', *Population Research and Policy Review* **28**(5), 551–567.

Jones, G., Steketee, R., Black, R., Bhutta, Z. & Morris, S. (2003), 'How many child deaths can we prevent this year?', *The Lancet* **362**, 65–71.

Kozlowski, J. & Stearns, S. (1989), 'Hypotheses for the production of excess zygotes: models of bet-hedging and selective abortion', *Evolution* **43**(7), 1369–1377.

Kraemer, S. (2000), 'The fragile male', *BMJ* **321**, 1609–1612.

Kramer, M., Lydon, J., Séguin, L., Goulet, L., Kahn, S., McNamara, H., Genest, J., Dassa, C., Chen, M., Sharma, S. et al. (2009), 'Stress pathways to spontaneous preterm birth: the role of stressors, psychological distress, and stress hormones', *American journal of epidemiology* **169**(11), 1319.

Lindstrom, D. & Berhanu, B. (1999), 'The impact of war, famine, and economic decline on marital fertility in Ethiopia', *Demography* **36**(2), 247–261.

Maconochie, N., Doyle, P., Prior, S. & Simmons, R. (2007), 'Risk factors for first trimester miscarriageresults from a UK-population-based case–control study', *BJOG: An International Journal of Obstetrics & Gynaecology* **114**(2), 170–186.

Macours, K. (2011), 'Increasing inequality and civil conflict in nepal', *Oxford Economic Papers* **63**(1), 1–26.

Martorell, R. & Habicht, J. (1986), *Growth in early childhood in developing countries*, Vol. 3, 2nd edn, Plenum Press, New York, pp. 241–262.

Meng, X. & Qian, N. (2009), 'The Long Term Consequences of Famine on Survivors: Evidence from a Unique Natural Experiment using China's Great Famine', *NBER Working Paper No. w14917* .

Menon, N. & Rodgers, Y. (2011), 'War and women's work: Evidence from the conflict in nepal', *Rutgers University. Mimeograph.* .

Miller, G. & Urdinola, B. (2010), 'Cyclicality, Mortality, and the Value of Time: The Case of Coffee Price Fluctuations and Child Survival in Colombia', *Journal of Political Economy* **118**(1).

MOHP, New Era and Macro International Inc. (2007), *Nepal Demographic and Health Survey 2006*, Ministry of Health and Population (Kathmandu, Nepal), New ERA, Macro International Inc., Kathmandu, Nepal.

MOHP, New Era and ORC Macro (2002), *Nepal Demographic and Health Survey 2001*, Family Health Division, Ministry of Health; New ERA; and ORC Macro, Calverton, Maryland, USA.

Mulder, E., Robles de Medina, P., Huizink, A., Van den Bergh, B., Buitelaar, J. & Visser, G. (2002), 'Prenatal maternal stress: effects on pregnancy and the (unborn) child', *Early human development* **70**(1-2), 3–14.

Murshed, S. & Gates, S. (2005), 'Spatial-horizontal inequality and the maoist insurgency in nepal', *Review of Development Economics* **9**(1), 121–134.

Nepomnaschy, P., Welch, K., McConnell, D., Low, B., Strassmann, B. & England, B. (2006), 'Cortisol levels and very early pregnancy loss in humans', *Proceedings of the National Academy of Sciences of the United States of America* **103**(10), 3938.

Nepomnaschy, P., Welch, K., McConnell, D., Strassmann, B. & England, B. (2004), 'Stress and female reproductive function: a study of daily variations in cortisol, gonadotrophins, and gonadal steroids in a rural Mayan population', *American Journal of Human Biology* **16**(5), 523–532.

Painter, R., Roseboom, T. & Bleker, O. (2005), 'Prenatal exposure to the Dutch famine and disease in later life: an overview', *Reproductive Toxicology* **20**(3), 345–352.

Rajab, K., Mohammad, A. & Mustafa, F. (2000), 'Incidence of spontaneous abortion in Bahrain before and after the Gulf War of 1991', *International Journal of Gynecology and Obstetrics* **68**(2), 139–144.

Roseboom, T., Van der Meulen, J., Ravelli, A., Osmond, C., Barker, D. & Bleker, O. (2001), 'Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview', *Molecular and Cellular Endocrinology* **185**(1-2), 93–98.

Rosenfeld, C. & Roberts, R. (2004), 'Maternal diet and other factors affecting offspring sex ratio: a review', *Biology of Reproduction* **71**(4), 1063–1070.

Samaraweera, Y. & Abeysena, C. (2010), 'Maternal sleep deprivation, sedentary lifestyle and cooking smoke: Risk factors for miscarriage: A case control study', *Australian and New Zealand Journal of Obstetrics and Gynaecology* **50**(4), 352–357.

Sandman, C., Glynn, L., Schetter, C., Wadhwa, P., Garite, T., Chicz-DeMet, A. & Hobel, C. (2006), 'Elevated maternal cortisol early in pregnancy predicts third trimester levels of placental corticotropin releasing hormone (CRH): priming the placental clock', *Peptides* **27**(6), 1457–1463.

Siega-Riz, A. M., Herrmann, T. S., Savitz, D. A. & Thorp, J. M. (2001), 'Frequency of eating during pregnancy and its effect on preterm delivery', *American journal of epidemiology* **153**(7), 647.

Stein, Z. & Susser, M. (1975), 'The Dutch famine, 1944-1945, and the reproductive process. I. Effects on six indices at birth', *Pediatric Research* **9**(2), 70–hyhen.

Strauss, J. & Thomas, D. (2008), *Health Over the Life Course.*, Vol. 4 of *Handbook of Development Economics*, Amsterdam: North Holland Press, chapter 54, pp. 3375–3474.

Symonds, M., Budge, H., Stephenson, T. & McMillen, I. (2001), 'Fetal endocrinology and development–manipulation and adaptation to long-term nutritional and environmental challenges', *Reproduction* **121**(6), 853.

Trivers, R. & Willard, D. (1973), 'Natural selection of parental ability to vary the sex ratio of offspring', *Science* **179**(4068), 90.

Ugwumadu, A. (2010), 'Chorioamnionitis and mid-trimester pregnancy loss', *Gynecologic and obstetric investigation* **70**(4), 281–285.

US Agency for International Development (2007), 'Nepal - humanitarian assistance fact sheet no. 1, june.', *http://pdf.usaid.gov/pdf_docs/PNADJ147.pdf . Accessed 5th May 2010* .

Valente, C. (2011), 'What Did the Maoists Ever Do For Us? Education and marriage of women exposed to civil conflict in Nepal', *Sheffield Economics Research Papers No. 2011009* .

Vonnahme, K., Hess, B., Hansen, T., McCormick, R., Rule, D., Moss, G., Murdoch, W., Nijland, M., Skinner, D., Nathanielsz, P. et al. (2003), 'Maternal undernutrition from early-to mid-gestation leads to growth retardation, cardiac ventricular hypertrophy, and increased liver weight in the fetal sheep', *Biology of reproduction* **69**(1), 133.

Wadhwa, P., Garite, T., Porto, M., Glynn, L., Chicz-DeMet, A., Dunkel-Schetter, C. & Sandman, C. (2004), 'Placental corticotropin-releasing hormone (CRH), spontaneous preterm birth, and fetal growth restriction: a prospective investigation', *American journal of obstetrics and gynecology* **191**(4), 1063–1069.

Wehby, G., Murray, J., Castilla, E., Lopez-Camelo, J. & Ohsfeldt, R. (2009), 'Quantile effects of prenatal care utilization on birth weight in argentina', *Health economics* **18**(11), 1307.

Wisborg, K., Barklin, A., Hedegaard, M. & Henriksen, T. (2008), 'Psychological stress during pregnancy and stillbirth: prospective study', *BJOG: An International Journal of Obstetrics & Gynaecology* **115**(7), 882–885.

Wooldridge, J. (2002), *Econometric analysis of cross section and panel data*, MIT press, Cambridge, MA, USA.

World Bank (2005), 'Nepal - resilience amidst conflict: an assessment of poverty in nepal, 1995-96 and 2003-04', *World Bank Report No. 34834-NP* .

Wynn, A. & Wynn, M. (1993), 'The effects of food shortage on human reproduction', *Nutrition and health (Berkhamsted, Hertfordshire)* **9**(1), 43.

A-1 Appendix Tables

Table A-1: Effect of Conflict Intensity on Antenatal Care

	(1) ANC Checks	(2) ANC Checks	(3) Iron/ Folic	(4) Iron/ Folic	(5) Tetanus Injection	(6) Tetanus Injection
Before conception	0.1766	0.2957	-0.0040	-0.0089	0.0781**	0.1404**
(total)	(0.1359)	(0.2031)	(0.0311)	(0.0519)	(0.0326)	(0.0533)
During pregnancy	2.3699		-0.2028		0.5881	
	(2.6372)		(0.7412)		(0.6069)	
First trimester		1.1372		0.4473		0.4545
		(2.7891)		(0.7810)		(0.6165)
Second trimester		-4.7722**		-0.7991		-0.9471
		(2.2389)		(0.7711)		(0.6183)
Third trimester		2.4411		0.1047		0.7222
		(3.1646)		(0.8069)		(0.7642)
Panel variable	District	District	District	District	District	District
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Maternal characteristics	No	Yes	No	Yes	No	Yes
Observations	7459	2646	7466	2648	7465	2648
Number of Groups	75	74	75	74	75	74
No. of clusters	75	74	75	74	75	74
R-squared	0.1250	0.2196	0.1915	0.2443	0.0957	0.1504
Mean Y_{idt}	1.907	1.526	0.371	0.322	0.598	0.549

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. Data collected only for the last birth. * p<0.10, ** p<0.05, *** p<0.01

Table A-2: Effect of Conflict Intensity on Help with Delivery

	(1) Trained Attdt	(2) No Help	(3) Trained Attdt	(4) No Help	(5) Trained Attdt	(6) No Help
Before conception	-0.0049	-0.0079	0.0130	-0.0049	0.0308	0.0164
(total)	(0.0170)	(0.0276)	(0.0172)	(0.0360)	(0.0304)	(0.0171)
During pregnancy	-0.1198	-0.4674				
	(0.3768)	(0.3314)				
First trimester			-0.1083	-0.5071	-0.1301	-0.5499**
			(0.2009)	(0.3240)	(0.3322)	(0.2147)
Second trimester			0.1969	0.1445	0.0604	0.0176
			(0.3117)	(0.1661)	(0.1616)	(0.1664)
Third trimester			0.1170	-0.2216	0.2691	0.3063**
			(0.2496)	(0.2613)	(0.3025)	(0.1494)
Panel variable	District	District	District	District	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Maternal characteristics	No	No	Yes	Yes	No	No
Observations	10598	10598	5630	5630	5630	5630
Number of Groups	75	75	74	74	2679	2679
No. of clusters	75	75	74	74	74	74
R-squared	0.0664	0.0359	0.1671	0.0364	0.0244	0.0178
Mean Y_{idt}	0.127	0.090	0.085	0.100	0.085	0.100

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. Data collected for all children under 5 years old. * p<0.10, ** p<0.05, *** p<0.01

Table A-3: Effect of Conflict Intensity on Immunization

	(1) Full Immun.	(2) No Immun.	(3) Full Immun.	(4) No Immun.	(5) Full Immun.	(6) No Immun.
Before conception	-0.0027	0.0097	-0.0224	0.0094	-0.0534	-0.0350*
(total)	(0.0422)	(0.0113)	(0.0542)	(0.0214)	(0.0628)	(0.0198)
During pregnancy	0.5354	-0.2081**	1.0102	-0.1988	-0.5142	-0.3347
	(0.5736)	(0.1037)	(0.6906)	(0.1870)	(0.7236)	(0.2288)
Since birth	0.6030	0.7371	0.8809	0.5999	-0.2569	-1.3293
	(2.2966)	(0.6351)	(2.9621)	(0.6514)	(2.6459)	(0.9683)
Age in months	0.0042***	0.0000	0.0058**	-0.0005	0.0036	0.0006
	(0.0015)	(0.0005)	(0.0024)	(0.0010)	(0.0033)	(0.0011)
Panel variable	District	District	District	District	Mother	Mother
Year of conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Maternal characteristics	No	No	Yes	Yes	No	No
Observations	7972	7972	4012	4012	4012	4012
Number of Groups	75	75	74	74	2556	2556
No. of clusters	75	75	74	74	74	74
R-squared	0.0475	0.0094	0.0838	0.0257	0.0133	0.0130
Mean Y_{idt}	0.747	0.017	0.717	0.023	0.717	0.023

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. Data collected for all children under 5 years old alive at the time of the survey, but restricted here to those aged 1 to 5 years old. * p<0.10, ** p<0.05, *** p<0.01

Table A-4: Short-run Effect of Conflict Intensity on Nutrition

	(1) Child WHZ	(2) Child WHZ<-2	(3) Mother BMI	(4) Mother BMI<18.5
Up to 3 months	-0.0771	0.0287**	-0.1911	0.0361
before interview	(0.0483)	(0.0143)	(0.1574)	(0.0223)
Last 3 months	-1.2043	0.9659	0.6934	0.6212
	(3.4749)	(0.8185)	(6.0132)	(0.9144)
Age in months	0.0000	-0.0016**		
	(0.0021)	(0.0008)		
Age in years			0.1620**	-0.0083
			(0.0644)	(0.0097)
Age in years squared			-0.0017	0.0000
			(0.0011)	(0.0002)
Panel variable	District	District	District	District
Year of conception FE	Yes	Yes	Yes	Yes
Calendar month FE	Yes	Yes	No	No
Month of Interview FE	Yes	Yes	Yes	Yes
Pregnancy characteristics	Yes	Yes	No	No
Maternal characteristics	Yes	Yes	Yes	Yes
Observations	9673	9673	6868	6887
Number of Groups	75	75	75	75
No. of clusters	75	75	75	75
R-squared	0.0575	0.0322	0.0602	0.0334
Mean Y_{idt}	-0.809	0.118	20.169	0.256

All regressions are estimated using the linear fixed-effect estimator. Pregnancy characteristics: as listed under Table 2. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. Columns (3) and (4) include only one observation per mother and exclude women who are pregnant at the time of the survey. * p<0.10, ** p<0.05, *** p<0.01

Table A-5: Short-run Effect of Conflict Intensity on Maternal Work

	(1) Work in last 7 days	(2) Work at home	(3) Off Work in last 7 days
Up to 3 months	0.0510*	-0.0303	-0.0109
before interview	(0.0268)	(0.0282)	(0.0192)
Last 3 months	-2.2543	-1.0135	3.5038**
	(1.3646)	(1.8361)	(1.3858)
=1 if pregnant	0.0067	0.0019	0.0139
	(0.0111)	(0.0144)	(0.0106)
Age in years	-0.0143	0.0197	0.0321***
	(0.0100)	(0.0136)	(0.0063)
Age in years squared	-0.0001	-0.0002	-0.0002*
	(0.0002)	(0.0001)	(0.0001)
Panel variable	District	District	District
Year of conception FE	Yes	Yes	Yes
Month of Interview FE	Yes	Yes	Yes
Maternal characteristics	Yes	Yes	Yes
Observations	7647	6226	6572
Number of Groups	75	75	75
No. of clusters	75	75	75
R-squared	0.1172	0.0355	0.0784
Mean Y_{idt}	0.814	0.150	0.062

All regressions are estimated using the linear fixed-effect estimator. Maternal characteristics: as listed under Table 3). District-correlated robust standard errors in parentheses. District casualties are expressed per 1000 inhabitants. Source: Author's calculations using Nepal DHS 2001 and DHS 2006. Samples include only one observation per mother. Column (2) is restricted to mothers who report working, and Column (3) is restricted to mothers who report having worked at some point in the 12 months preceding the survey. * p<0.10, ** p<0.05, *** p<0.01

A-2 Pathways from Violent Conflict to Fetal and Child Health

The potential adverse effects of violent conflict on child health are numerous: direct effect of violence, indirect effects through destruction of sources of income and crops, looting, racketeering, disease prevalence, reduced investments in (health) human capital due to a reduction in life expectancy, destruction of health care facilities.

The expected adverse effect of these on nutritional status of mothers and children is clear, although the evidence is at present still limited to the few studies reviewed in Section 1, which have found that exposure to civil conflict decreases children's long-term nutritional status as captured by height-for-age.

The transmission of maternal health to fetal and child health deserves more discussion. Fetal loss is by no means a negligible phenomenon: in clinical studies in developed countries, 12-15 percent of clinical pregnancies (i.e., more than 6 weeks after the last menstrual period) end in a miscarriage, much more if pre-clinical pregnancies are included, whilst typically less than one percent end in a still birth (Garcia-Enguidanos et al. (2002); Cnattingius et al. (1998); Cai & Feng (2005); Nepomnaschy et al. (2006)). The causes of miscarriages and still births are not well understood (Incerpi et al. (1998); Garcia-Enguidanos et al. (2002); Cai & Feng (2005); Wisborg et al. (2008)), with only two overwhelmingly acknowledged causes: chromosomal anomalies and intrauterine defects (Garcia-Enguidanos et al. 2002). From an evolutionary point of view, the high rate of fetal loss in human reproduction may be seen as a mechanism leading to natural selection of the fittest (Kozlowski & Stearns (1989); Nepomnaschy et al. (2006)), and so an increased rate of fetal loss due to civil conflict, if it is observed, may contribute to dampening the adverse effects of the conflict on the population of children effectively born. Although miscarriages in early and late pregnancy are often studied together, it is generally accepted that chromosomal abnormalities matter more for early- than for late miscarriages, whilst environmental factors and infections matter more for late miscarriages than for early ones (Ancel et al. (2000); Allanson et al. (2010); Samaraweera & Abeysena (2010);Ugwumadu (2010)).

There are four main pathways through which violent conflict during pregnancy could affect fetal and child health, namely: use of health care, maternal nutrition, maternal stress, and maternal work. In observational studies, all these variables are likely to be correlated with other determinants of child health, and randomization is generally not feasible. Therefore, the causal nature of the correlations reported in the literature is often unclear.

In the case of antenatal care, it is undeniable that some health inputs such as maternal tetanus toxoid immunization and delivery by a skilled birth attendant prevents neonatal deaths (Jones et al. 2003). In addition, instrumental variables of varying reliability have been used (e.g., marital status, maternal employment, health care availability in the mother's area), and findings overall suggest that early initiation of antenatal care and the quantity of antenatal care improve birth weight (see Wehby et al. (2009) for a recent example).

Maternal nutrition has been linked conclusively to abnormal placental growth in animals (Symonds et al. (2001), Bispham et al. (2003), Vonnahme et al. (2003)). In humans, studies controlling for a number of confounders have found that poor maternal nutrition adversely affects indicators of fetal and infant health such as pre-term delivery, birth weight, miscarriage, still birth and infant mortality (Herrmann et al. (2001); Siega-Riz

et al. (2001); Helgstrand & Andersen (2005); Maconochie et al. (2007)). Studies focusing on the impact of *in utero* exposure to famines have also found a decrease in birth weight, and an increase in infant mortality and miscarriage, although the evidence on miscarriage, in particular, is mostly suggestive. [15] Recently, Almond & Mazumder (2008) use the exogenous variation in the timing of the holy month of Ramadan to estimate the impact of *in utero* exposure to fasting on the health outcomes of Arab children and find that it decreases birth weight.

The effect of maternal stress in animals on birth outcomes is well established (Mulder et al. 2002). The findings from (non-experimental) studies on humans are less consensual, but there is consistent biomedical evidence of the adverse effect of maternal stress on pre-term delivery (Austin & Leader 2000), whilst the effect on other pregnancy outcomes such as birth weight is less robust (Austin & Leader (2000); Mulder et al. (2002)).[16] A somewhat dissonant finding in the economics literature is that of Camacho (2007), who finds that the occurrence of landmine explosions in Colombia has a small but significant effect on birth weight, but that the effect on low birth weight and pre-term delivery is not robust to controlling for maternal fixed-effects. Rajab et al. (2000) compare the incidence of spontaneous abortions in the 5 years before and the 5 years after the Gulf War based on hospital records from the main referral hospital of Bahrain, and find an increase in referrals in the post-war period. However, confounding factors that increase the occurrence of fetal loss and/or referral to the main hospital in the country over the 10-year period centered around the war cannot be ruled out. Wisborg et al. (2008) have also found that women who report experiencing a high level of psychological stress around 30 weeks of gestation are 80 percent more likely to suffer a still birth. Few studies shed light on whether and how the timing of maternal stress matters. Overall, it would appear that maternal stress has a more pronounced effect on pre-term delivery when mothers are exposed to stress early in pregnancy, but the effect on other pregnancy outcomes is unclear.[17]

[15] Cai & Feng (2005) estimate that the famine caused by the Great Leap Forward increased fetal loss. These authors use Chinese data on retrospective fertility histories to estimate logistic regressions of miscarriage and still birth on a set of controls including a continuous year variable and dummies for the years where crises occurred, namely 1958-62 and 1966-68. The coefficients on the interaction between the year dummies and socioeconomic characteristics are used to support the authors' claim that the year effects are linked to the famine of 1959-1961 and "the most intense year" of the Chinese Cultural Revolution, 1967 (Cai & Feng (2005), p.310). Nonetheless, in the absence of within-country variation in exposure to the shock considered, and without comprehensive controls for time-varying unobservables, it is hard to interpret the estimated effects as causal. Wynn & Wynn (1993) offers graphical evidence of a sharp decrease in fertility and birth weight as well as of an increase in miscarriages in a number of German cities in the immediate post-war, which the authors attribute mostly to food shortages. Stein & Susser (1975), Roseboom et al. (2001), and Painter et al. (2005) find that exposure to the Dutch famine in the third trimester of gestation decreased weight and length at birth, and increased the likelihood of infant mortality, whilst the effect of exposure during the first and second trimester are smaller, which Roseboom et al. (2001) suggest may be due to increased fetal loss.

[16] The biological transmission of maternal stress to the fetus is not yet fully understood (Kramer et al. 2009), but the main mechanism suggested in the literature is an increased placental Corticotrophin-Releasing Hormone (CRH) around weeks 30-33 of gestation (Mulder et al. (2002); Sandman et al. (2006); Wadhwa et al. (2004)). Two other much less studied suggested mechanisms are (i) that of a reduced utero-placental blood flow due to the activation of the sympathetic nervous system, and which may contribute to fetal growth restriction (Mulder et al. 2002) and (ii) the hypothesis that stress may weaken the maternal immune system, thereby increasing the likelihood of infection, which, in turn, are known to be one of the major risk factors for pre-term birth (Hobel & Culhane 2003).

[17] Glynn et al. (2001) found that, in their sample of 40 Californian women who were pregnant during the 1994 earthquake, the reduction in gestational length increased the *earlier* in the pregnancy the

Another mechanism through which violent conflict may affect fetal health would arise if parents, and mothers in particular, were less likely to work when there is more fighting taking place. A growing body of literature on the impact of economic downturns on child health suggests that the empirical link between these two variables is context-specific (Miller & Urdinola 2010). On the one hand, a slow down in economic activity deteriorates the household's ability to afford health care, both for expecting mothers and young children. On the other hand, some health inputs are likely to be costly in terms of time more than in financial terms, so that health inputs may increase when the opportunity cost of time decreases (Miller & Urdinola 2010). In addition, a decrease in work-related strenuous activities and a reduction in exposure to pesticides in the predominantly agricultural society of Nepal might reduce the occurrence of miscarriages and still births (El Metwalli et al. (2001); Figa-Talamanca (1984)), which could *increase* the frailty of the average born child.

Finally, fetal and child health may be affected through selection on parental characteristics. The direction of this selection mechanism is undetermined *a priori*. On the one hand, poorer, less educated parents may be less likely to delay fertility to avoid times of unrest, e.g. due to lower cultural acceptance of contraceptive methods or less appropriate use of methods, which would tend to worsen the health of the average child. On the other hand, better-off parents may be more sheltered from the adverse consequences of conflict and thus be less prone to taking into account conflict-related violence in their fertility decisions.

Civil conflict may affect not only fertility decisions, but also reproductive ability, through the adverse impact of either malnutrition or stress (Mulder et al. (2002); Nepomnaschy et al. (2004); Painter et al. (2005)). Demographic studies have reported a drop in fertility during wartime (Lindstrom & Berhanu (1999);Eloundou-Enyegue et al. (2000); Agadjanian & Prata (2002); Jayaraman et al. (2009)). In particular, a study by Agadjanian & Prata (2002) has shown that better educated and wealthier women were more likely to decrease fertility during post-electoral violence in Angola in the 1990s.

In summary, (i) a decrease in the use of prenatal care and clean delivery is likely to decrease birth weight and increase neonatal mortality; (ii) the effect of undernutrition on fetal loss is unclear, but acute maternal malnutrition in late gestation tends to decrease birth weight and increases infant mortality; (iii) prenatal psychological stress is likely to increase the likelihood of pre-term delivery, especially when it occurs in the first 20 weeks of gestation, and maternal stress experienced at any stage of pregnancy may lead to fetal loss or other adverse infant health outcomes; finally, (iv) the effect of a reduction in maternal work on fetal growth and child health is undetermined *a priori*.

---

earthquake took place. In addition, the link between maternal stress and heightened CRH levels has only been established up to early- to mid- second trimester: Hobel et al. (1999) and Sandman et al. (2006) find maternal stress at respectively 18-20 and 15 weeks of gestation to be correlated with placental CRH levels in the early third trimester, which have in turn been shown to predict pre-term delivery (Wadhwa et al. 2004).