Surviving the Genocide: Child Mortality and the Rwandan Genocide

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Abstract

We investigate the relationship between the 1994 Rwandan genocide and children's mortality. We combine birth history data drawn from the 2000 Rwanda Demographic and Health Survey with prefecture-level information on the intensity of the conflict. Considering both *in utero* and postnatal war exposure, our estimates from discrete time proportional hazard models reveal large positive association between the exposure to the conflict and infant and child mortality. For infant mortality, these effects are associated to residence in districts with a high intensity of war and to bereavement in the family during the genocide. For children who survived, the main effect stems from having lived either in life or *in utero* during the conflict, with an increase in the hazard of mortality by around 10 percentage points per month of exposure. The same effect holds even if exposure was only *in utero*. This might represent a helpful indication to identify the group of more fragile children to target with policy actions aimed at improving child health after a war.

JEL Classification: I20, J13, O12, Z13

Keywords: genocide, child mortality, child health, survival analysis, Rwanda

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I. Introduction

> *"Many of today's armed conflicts take place in some of the world's poorest countries, where children are already vulnerable to malnutrition and disease, and the onset of armed conflict increases death rates up to 24 times. All children are at risk when conflicts break out, but the most vulnerable are those who are under five and already malnourished"* (Machel, 1996).

This paper investigates the relationship between civil war and children's mortality. The study of the consequences of wars for children is part of a more general field of research that aims at establishing a causality nexus between early living conditions and outcomes later in life. Negative shocks affecting newly born children's health may lead to lower height, less cognitive achievement, slower human capital accumulation, lower productivity and wages as adults, particularly in low income countries (Strauss and Thomas 1998). Exploiting the temporal and geographic variation of exogenous shocks, some recent papers provide evidence of causal links between, for example, crop failure (Banerjee 2010 et al.), famine (Meng and Qian 2009), and rainfall (Maccini and Yang 2009), with children's outcomes later in life. Negative shocks exclusively experienced while *in utero* have also been proven to affect children's health (Stein 1975; Almond 2006).

Turning to the focus of this paper, some studies have analyzed the impact of war on children's outcomes several years after the end of a conflict. As far as health and human capital are concerned, some studies find a negative impact of war on children's height for age z-scores, while others on children's schooling. Bundervoet et al. (2009), for example, finds that an additional month of war exposure decreases children's height for age z-scores by 0.047 standard deviations compared to non-exposed children in Burundi. Akresh et al. (2011) shows that in poor and non-poor households in Rwanda, boys and girls born during the conflict in regions experiencing fighting are negatively

impacted with height for age z-scores 1.05 standard deviations lower. Akresh et al. (2012) finds that individuals exposed to the Nigerian civil war of 1967-70 at all ages between birth and adolescence exhibit reduced adult stature and these impacts are largest in adolescence.

As far as schooling is concerned, Akresh and De Walque (2010), for example, finds a strong negative impact of Rwanda's genocide on children's schooling, with exposed children completing half a year less education, which amounts to an 18.3 per cent decline in school completion. Shemyakina (2011) finds that exposure to violent conflict has a large and statistically significant negative effect on the enrolment of girls (not of boys) in Tajikistan. León (2012) finds that in Peru, exposure to violence reduces adult education by 0.31 years, and in the short-term the effects are stronger than in the long run.

Surprisingly enough, although child mortality is of interest to economists for many reasons (Currie, 2008), in this growing body of literature little attention has been paid to the survival of children exposed to a conflict. In this paper, we address the issue of whether a war, beyond its immediate consequences on mortality rates, may continue to generate disruptive effects on child health and mortality, for a long time after its conclusion. In other words, we ask whether a war may leave behind a "permanently fragile" generation composed by the children who have been exposed to it.

A positive answer to this question has relevant policy implications, since aid intervention, in such case, should not only face the war and its direct post-war consequences, but also plan middle-term targeted actions to support the fragile generation. The only paper that attempts at answering a similar question is that by Verwimp (2011), who finds a positive effect on child mortality of child undernutrition experienced during civil war in Burundi.

We study the case of Rwanda. Using the birth history data drawn from the 2000 Rwandan Demographic and Health Survey (RDHS 2001), we investigate the impact of the 1994 Rwandan genocide on mortality of the cohorts of children who were exposed to it, both *in utero* and in life. From a methodological point of view, the Rwandan case is particularly suitable for such analysis

since the fast recovery of the population - dramatically reduced by the war - to its initial dimension, might induce to suppose that the war, after all, did not generate any lasting consequences. We question this supposition.

We focus both on infant mortality (mortality in the first year of life) and on child mortality (mortality in the first five years of life). Exploiting temporal and spatial variation, we devise a battery of indicators to capture exposure to the genocide. We define exposure as having lived in the war period and use the variable "length of exposure in months" as proxy. Further, using information on the geographic intensity of the genocide contained in Justino and Verwimp (2008), a child's residence is classified to be in a prefecture with "very intense", "moderately intense" and "not intense" genocide. Finally, genocide intensity is also measured with mother's bereavement, with the ratio of deaths among maternal siblings in 1994.

We estimate discrete-time survival models with piecewise constant baseline hazards, and, in order to disentangle the long-run effects of the war, we estimate separate models for children who were exposed and might have died during the conflict, and children who survived it. To test the assumption of whether pre-natal exposure might lead to increased child mortality, we also estimate the model for the subsample of children who were only exposed while *in utero*.

We find large positive effects of exposure to the conflict on infant and child mortality. For the survivors, we find that child mortality is significantly correlated to war exposure, a result that shows that the effects of the conflict last several years after the end of the war.

The paper is structured as follows. Section II briefly introduces the Rwandan genocide. Section III describes the data end the econometric method used. Section IV presents the estimation strategy. Sections V and VI discuss the results for infant mortality and child mortality respectively. Section VII summarizes and concludes.

II. The Rwandan Genocide

The African Great Lakes region has been disrupted by civil and inter-state wars, genocides and coups d'état since the mid-nineties. Only nowadays, it is possible to glimpse a feasible path of stabilization of the region even if armed conflicts are still standing (for example in the Kivu region in the Democratic Republic of Congo) and many states in the region are still fragile. In this context, Rwanda played a crucial role: despite its small dimension, Rwanda contributed to destabilize the whole region. In 1994, Rwanda experienced a genocide whose inhuman fierceness can be found in very few other circumstances in human history: in only 100 days (in the period ranging from April to July 1994) between 500,000 and 800,000[1] Tutsi and moderate Hutu[2] people were killed by the Rwandan army (Force Armées Rwandaises, FAR), by the police and by the members of the Interahamwe militia.[3] As yet, there is no consensus about the estimated number of deaths, but the total number, including those indirectly due to the genocide (for example deaths occurred among displaced people), ranges from 800,000 (UN) to one million (Rwandan Government). Despite this sudden and tremendous population loss, Rwanda quickly went back to the pre-genocide path of demographic growth (see Figure 1). This recovery, however, does not exclude the existence of long-term negative consequences of the genocide for the Rwandan population.

[1] These estimates are provided in the report "Rwanda: the Preventable Genocide" (OAU, 1999).
[2] Hutus that were not supporters of the "Hutu power" ideology.
[3] Detailed data and information concerning Rwandan genocide are available on the website www.genodynamics.com.

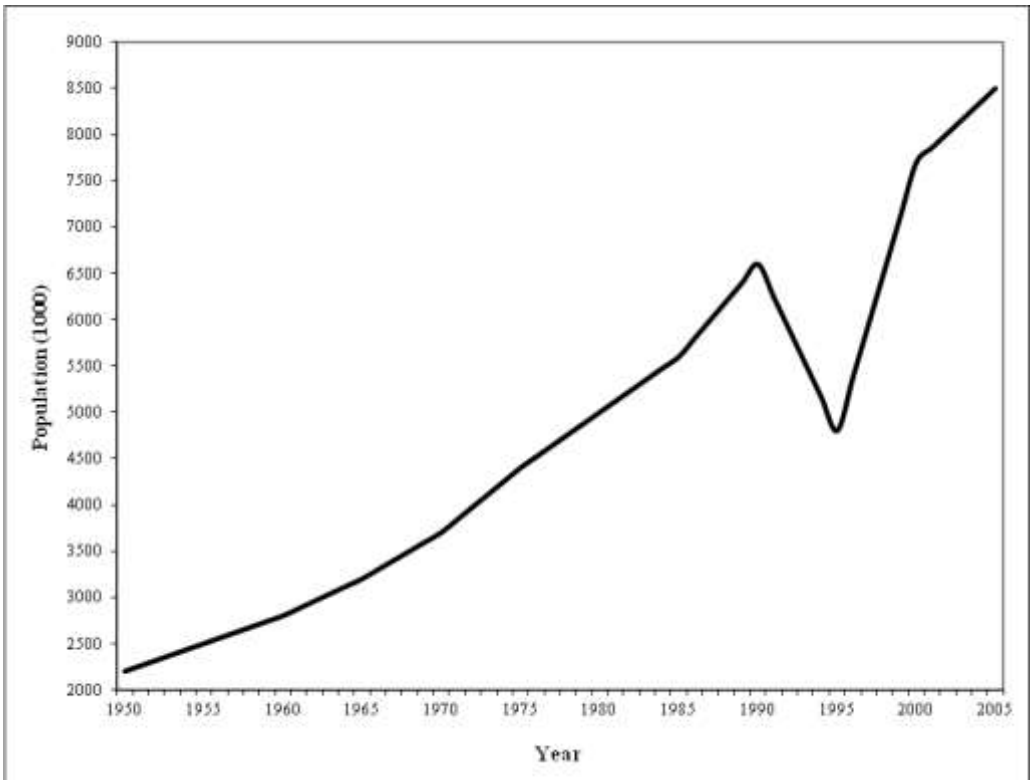

Figure 1.

Trend of Rwandan Population (1950-2005)

Source: World Population Prospects: The 2008 Revision (UN Population Division, 2008) as reported in Hong et al. (2009)

The Rwandan genocide results from the combination of exogenous and endogenous factors. Newbury (1998) observes that ethnic tensions are a constant of the Rwandan history and that they have been managed in various ways, including violence. Exogenous factors (chiefly the Belgian colonization and the Belgian "indirect rule" system) have induced a calcification of ethnic identities and consequently reduced the number of viable solutions of ethnic tensions. Other authors stress the role played by economic factors such as the austerity measures imposed by the IMF and The World Bank (Chossudowsky 1996), and the sharp fall of the price of coffee (Verwimp, 2003).

The deaths directly due to violence of the genocide were not randomly distributed among the Rwandan population. In fact, there was a quite well defined targeting: the surplus mortality directly

due to the genocide was particularly high among urbanized Tutsi[4] adult males, who were richer and more educated than the average of the population (De Walque and Verwimp, 2009). Since children were not an explicit target of the violence committed during the genocide, it becomes relevant to analyze the consequences of the events occurred between April and July 1994 for child mortality.

III. Data and Econometric Specification

Following the current definitions of child mortality (Van der Klaaw and Wang, 2011) we focus on "infant mortality" (child mortality in the first year of life) and on "child mortality" (child mortality in the first five years of life). We use the 2000 Rwandan Demographic and Health Survey (RDHS 2001)[5] which records each woman's "birth history" collecting recall information on each birth in her lifetime from January 1990 (the first available date of birth in the survey) to the time of the interview.[6] In addition, RDHS collects information on several aspects of child health, such as anthropometrics, vaccination, prenatal and delivery assistance. However, this information is only available for children who were born no later than five years before the interview (that is, at most in 1995). Unfortunately, since our focus is on children born around the period April-July 1994, we cannot investigate these aspects.

Information on children's month and year of birth - and month and year of death in case of children who are not alive at the time of the interview - allows us studying child survival probability with monthly data. Given this data structure, we treat children's survival histories as formed by monthly intervals, and estimate a discrete time specification.[7] Measuring time in monthly intervals *j* indexed

[4] It must be noticed that even if the mortality rate were higher among Tutsi, the most part of victims (in absolute numbers) probably were Hutu.
[5] RDHS 2000 is the first available survey after the genocide. Since recall information on birth history is collected only for the past five years, we cannot use also the more recent 2005 and 2010 DHS surveys for our analysis.
[6] The interviews took place between June 26 and November 30, 2000.
[7] To estimate the model we need to transform the data in person-periods, which means having for each child in the data matrix as many rows as the months at risk.

by the positive integers, we choose a complementary log log functional form (Jenkins 1995) [8] to model the *jth* hazard rate for each child's survival up to month *j* as follows:

$$h(j, X) = 1 - \exp[-\exp(\beta' X + \gamma_j)] \qquad (1)$$

where *h(j,X)* is the discrete time hazard function, *X* is a vector of household, maternal, and child characteristics. It also includes several variables approximating the exposure to civil war. γ_j is the baseline hazard for interval *j*.

Another crucial point is the specification of a functional form of the baseline hazard. We use a piecewise constant specification assuming, in the model for infant mortality, a constant baseline hazard for each month. For the child mortality model, we group months in each constant baseline hazard according to what is common knowledge about the timing of mortality (higher in the first month and in the first year than in the subsequent years). Under these assumptions, we express γ_j in (1) as follows:

$$\gamma_j = \gamma_1 D_1 + \gamma_2 D_2 ... + \gamma_T D_T \qquad (2)$$

where *T* is the number of interval groups and the *Ds* are the dummies for each group. Using a specification without the constant term, we end up with twelve monthly dummies in the infant mortality model and with four dummies for months 5 to 12, 13 to 36, 37 to 48 and 49 to 60 in the child mortality model. In each model we control for "frailty", that is, unobservable heterogeneity. Moreover, since DHS surveys have a stratified structure (individuals are nested into households, households into communities and communities into regions) we choose to compute clustered standard errors, allowing for intra-group correlation. Finally, we have computed weighted

[8] The *cloglog* model is the discrete time representation of a proportional hazards model.

regressions, using DHS sample weights to take account of the sample selection probability of each household.

IV Estimation Strategy and Genocide Variables

We estimate three model specifications. In the first one, we analyze the consequences of the genocide for infant mortality of children who were exposed to it.

We then turn to the main question this paper, regarding the long-term effects of the genocide, and focus on the mortality of children who survived it. We aim at comparing the mortality of the survivors, with the mortality of children conceived and born after the genocide. It is reasonable to assume that both the survivors and children who were conceived right after the genocide have experienced similar post-genocide conditions (aid intervention and the like), so that the unobserved heterogeneity bias due to changes in health services in the observed time period may be considered as limited.

Given this caveat, for the second model specification, we select: 1) children who were exposed to the genocide - *in utero*, in life or both - and who survived it; 2) children who were born after the genocide, were not exposed to it while in utero, and could turn five before the date of the interview. In the third model we restrict the latter sample selecting, among the survivors, only children who were exposed while *in utero.*

To estimate infant mortality (model 1), we select the cohorts of children who were exposed to the genocide during their first year of life, namely, those born between May 1993 and July 1994, and also those exposed to the genocide while *in utero*, born between August 1994 and April 1995 (2,889 children in total).[9]

For child mortality of children who survived the genocide or were conceived after it (model 2),we select all children born after March 1994, who survived the first four months of life (the duration of

[9] As data concerning premature delivery are not available, we have imputed a standard length of pregnancies of 9 months.

the war).[10] Moreover, we drop children who were not potentially able to turn five before the month of mother's interview in 2000 – whether alive or not at the time of the interview. This sample consists of 2,099 children (both exposed and unexposed).

In order to test whether the mere pre-natal exposure has an effect on mortality, we also estimate the model on the subsample of children born after August 1994 who were just exposed while *in utero* plus the group of those who were conceived and born after the genocide (model 3), a sample amounting to 1,442 children.

As for the genocide variables, we approximate the intensity of exposure exploiting the variation of the genocide at the mother/household and geographic levels with two variables, the *ratio of deaths among maternal siblings* and *prefecture genocide intensity*. The variable *ratio of deaths among maternal siblings,* drawn from a special section of RDHS (2001) on siblings' mortality data, is the ratio of the number of deaths in 1994 among siblings of women aged 15-49. This variable takes account of the level of stress faced by the mother/household and of the weakening of the household social network due to the genocide (De Walque and Verwimp 2009).

The prefecture genocide intensity is a dummy variable constructed using the Justino and Verwimp (2008, Table 1b) classification of prefectures by genocide intensity into *Prefectures with very intense genocide* (Butare, Cyangugu, Gikingoro and Kibuye), *Prefectures with moderately intense genocide* (Gitarama, Kigali and Kibungo) and *Prefectures with not intense genocide* (Byumba, Giseny and Ruhengeri).[11] This proxy, which is built using the prefecture of residence of the household in 2000, captures exposure intensity only for households that either did not move from the prefecture after the genocide or returned afterwards. For households that have permanently changed residence after the genocide, this proxy does not have the intended meaning. This is a disadvantage, since, as previously discussed, displacement is another widespread dramatic consequence of a conflict. In order to control

[10] This sampling rule requires that children born in April 1994 survive for four months until the end of the war. For this reason, all children in this sample, both exposed and unexposed, must have survived for four months.

[11] The classification reflects the percentage (high, medium and low) of Tutsi at the provincial level before the genocide.

for the possible displacement of the household during the genocide, we use the information on the mothers' number of years of residence in the place where she is residing at the time of the interview.[12] If the mother declares to have been living in the current place of residence since 1994, we build a dummy which equals one if *Mother changed prefecture of residence during the genocide*.

In Model 2 and 3 a more informative variable has been used to approximate the individual intensity of exposure to the genocide, namely, *Number of months of exposure*. It is a variable ranging from 0 to 4, indicating the number of months of exposure to the genocide both during the first year of life and *in utero*. The use of this variable in the infant mortality model would be misleading. In fact, since the first months of life are physiologically characterized by the highest probability of death, as months of exposure to the genocide increase, also the probability of child survival increases. This effect dominates the negative effect of the genocide, and the variable *Number of months of exposure* turns out to be positively correlated with child survival. Since our child mortality analysis focuses on the survival after the fourth month of life, this problem does not arise.

As for the other control variables, we use the classic control variables for infant and child mortality such as birth spacing, mother's education, mother's age at birth, household wealth and so on (see, for example, Van der Klaauw and Wang 2011). We illustrate the most relevant ones.

Oral rehydration therapy is considered as a pivotal component of the struggle to reduce infant mortality, as it is an effective, cheap and easy to use mean to mitigate the effect of diarrhea and other gastro-enteric diseases (Victora et al. 2000). We include a dummy for mother's knowledge of oral rehydration therapy.

Given the African context, it would be relevant to control for HIV prevalence among mothers and fathers. Rwanda is characterized by around 3 per cent HIV prevalence (source UNAIDS, 2013). Unfortunately, this information is not present in the 2000 DHS survey. In order to investigate the

[12] In the women questionnaire it is asked since how many years the interviewee has been residing in the current place of residence.

relationship between women's empowerment and child mortality, contraception is introduced in the model as a proxy of women's autonomy and emancipation (Eswaran 2002). A dummy variable equal to one if child's mother has ever used a modern contraceptive method is therefore included in the models.

The association between mothers' education and children's health is one of the most investigated and verified relationships (see, for example, Mosley and Chen 1982), and we include a dummy for mother's literacy.[13] Mother's age at birth is another important aspect possibly related to child health. In fact, health and development may be different between children of teenage or very old mothers and normal age mothers (Rothenberg and Varga, 1981).[14] Mother's age at birth is included as a continuous variable with a quadratic specification.

Birth spacing is very important for mothers' and children's health. It may proxy mothers' fertility preferences at the time of the child's conception, since close births tend to put mothers' physical condition under pressure and children born in this condition tend to be more fragile (Winikoff 1983). To control for this, a categorical variable concerning birth interval (with the class 1-23 months as reference category) has been introduced.

We control for sex of the child and sex of the household head including two gender dummies (equal to one if the child is male and if the household head is female).

The effect of wealth is caught by the scores of the wealth index computed according to the methodology described by Filmer and Pritchett (2001) and Rutstein (2008). Infant and child mortality are usually negatively correlated with the household wealth index. As one of the main sources of contamination is water, the presence in the household of an improved source of water is

[13] The DHS survey includes variables describing the interviewed women's level of education. However, some incongruity in the data is to be noticed: 723 women (9.5% of total educated women) who declared to have reached at least the degree of primary education are, however, unable to read. We therefore prefer to use a dummy variable for literacy, as literacy is checked by the interviewer with a quick test.

[14] The authors find, for the US, that older maternal age has an adverse effect on a child's educational outcome regardless of whether other factors are controlled for or not. Instead, the association of young maternal age and long-term morbidity is not significant when controlling for other factors.

an important factor to improve child health.[15] A dummy variable indicating whether a household has access to an improved water source is included in the models.

Table 1 summarizes the characteristics of the estimated models.

Table 1

Synopsis of the Estimated Complementary Log Log Models

Estimated Models	Children's type of exposure/non exposure	Birth cohorts	Variables of exposure to the genocide
Model 1 Infant Mortality (1-12 months)	In life and *in utero* exposure.	Children born between May 1993 and April 1995.	*Ratio of deaths among maternal siblings in 1994. Prefectures with very intense genocide Prefectures with moderately intense genocide* (baseline: *prefectures with not intense genocide*). *Mother changed prefecture of residence during the genocide (*baseline: *Mother did not change).*
Model 2 Child Mortality (4-60 months)	Exposed *in utero* and/or in life survivors. Unexposed conceived after the genocide.	Children born after March 1994 who could turn five before or at the date of the interview	*Ratio of deaths among maternal siblings in 1994.* P*refectures with very intense genocide Prefectures with moderately intense genocide* (baseline: *prefectures with not intense genocide*). *Mother changed prefecture of residence during the genocide (*baseline: *Mother did not change). Number of months of exposure*
Model 3 Child Mortality (4-60 months)	Exposed *in utero* survivors. Unexposed conceived after the genocide.	Children born after August 1994 who could turn five before or at the date of the interview	*Ratio of deaths among maternal siblings in 1994.* P*refectures with very intense genocide Prefectures with moderately intense genocide* (baseline: *prefectures with not intense genocide*). *Mother changed prefecture of residence during the genocide (*baseline: *Mother did not change). Number of months of exposure*

V. Infant mortality: Results

To start with, we present some suggestive graphical non-parametric evidence on the relationship between the genocide and infant mortality. To this end, we compare infant mortality of the exposed

[15] The definition of improved water source is taken from DHS (2008). According to this definition, public taps, protected private and public wells and piped water are considered improved water sources.

children, with infant mortality of the cohorts born one year before (between May 1991 and April 1993, 2,579 children) and during the year following the genocide (between May 1995 and April 1997; 3,271 children).[16]

Figure 2 shows that the survival curve of children belonging to the generation exposed to the genocide lies under the survival curves of the unexposed groups. Moreover, the log-rank test for the equality of survival curves of the exposed and unexposed groups indicates that they differ significantly from each other.

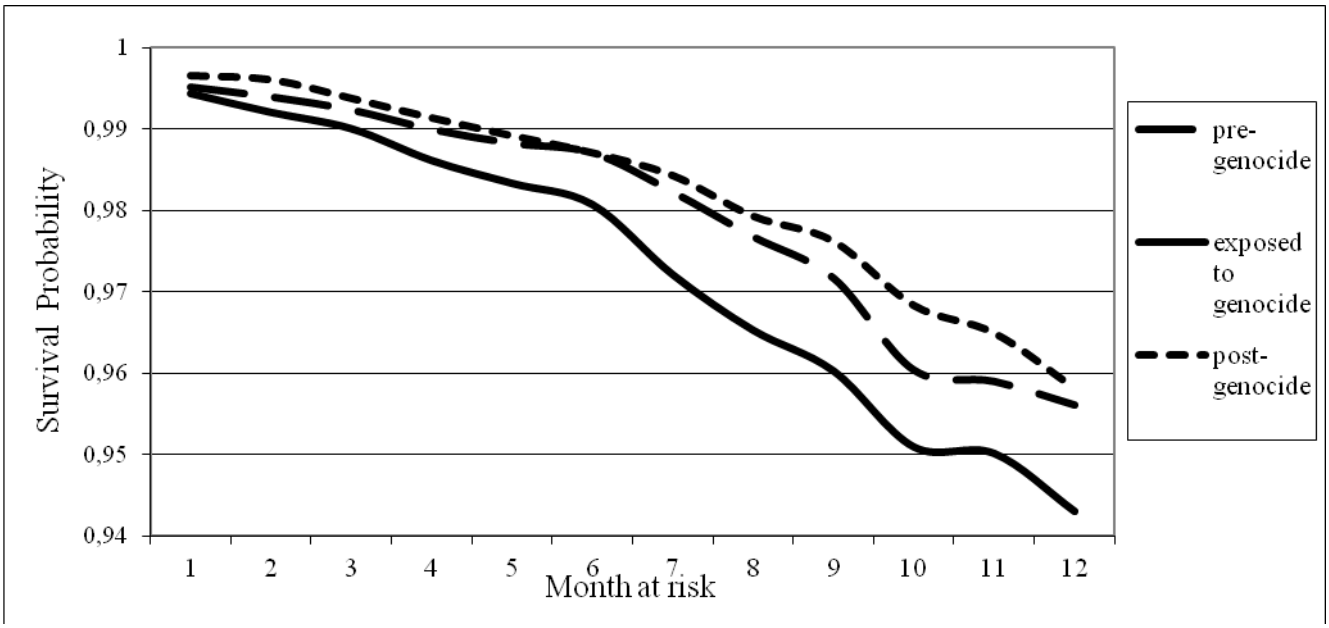

Figure 2

Infant mortality. Survival Curves for Exposed and Unexposed Children

Note: log-rank test for equality of survival functions chi2(2)=80.74; Pr>chi2= 0.00

Indeed, the increase in infant health of the post-genocide cohorts might have been driven by a positive time trend, or by the international aid and post-war policies targeting infants and children

[16] Note that no survival spell at the child level is either left or right censored since, in order to have a balanced sample, we have sampled all children born up to two years before and up to two years after the genocide. Since the last post-genocide children in the sample were born in April 1997 and the interview period ranged from June 26th to November 30th of year 2000, all sampled children could survive one year.

after the genocide. The pre-genocide group, however, shows a higher survival rate as well, thus confirming the relevance of our question.

In our model estimation we focus on the exposed birth cohorts. Figure 3 shows the structure of exposure to the genocide for each monthly cohort of children distinguishing between postnatal and *in utero* exposure. For example, the 138 children born in June 1994 were directly exposed to the genocide for two months and *in utero* for another two months.

Figure 3 *Structure of Exposure to the Genocide and Infant Mortality by Monthly Birth Cohort*

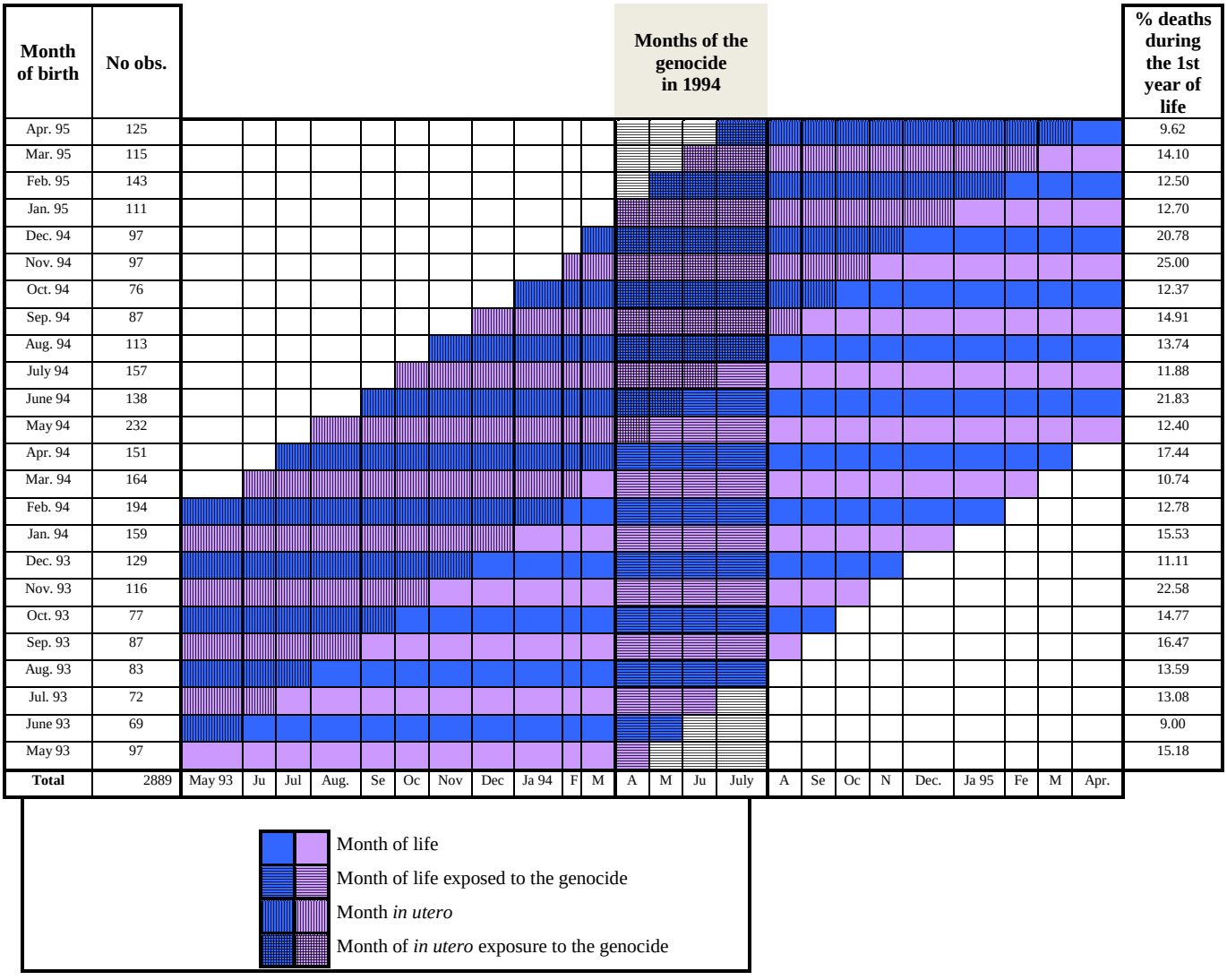

Table 1 presents the descriptive statistics of the model.

Table 1

Descriptive Statistics for Infant Mortality

	Infant mortality	
Variables	Model 1	
	Mean	S.D
Genocide intensity		
Ratio of deaths among mat. siblings 1994	0.11	0.21
Prefectures with not intense genocide	0.32	0.47
Prefectures with moderate intensity	0.35	0.48
Prefectures with high intensity	0.32	0.47
Mother changed pref. of residence .	0.10	0.30
Household characteristics		
Urban	0.18	0.38
Wealth index score	-0.10	0.87
Access to improved water source	0.80	0.40
Household head is female	0.34	0.47
Maternal characteristics		
Mother knows oral rehydration	0.88	0.33
Mother used contraception at least once	0.27	0.45
Mother can read	0.57	0.50
Child characteristics		
Mother's age at birth	28.75	6.51
First-born	0.20	0.40
1-23 months since previous birth	0.27	0.44
24-35 months since previous birth	0.26	0.44
36 or more since previous birth	0.47	0.50
Male	0.50	0.50
Number of observations	2,889	
Months of birth	05/1993-04/1995	

The results of the parametric *cloglog* duration analysis of infant mortality are presented in Table 2. The estimated coefficients measure the hazard of dying during the first year of life.

Table 2 *Infant Mortality of the Exposed Children: Complementary Log Log regression*

	Coefficients	Exponentiated Coefficients
Exposure to genocide		
Ratio of deaths among mat. siblings in 1994	0.631***	1.88
	(3.23)	
Mother changed prefecture of residence during the genocide	-0.226	0.797
	(0.204)	
Prefectures with moderately intense genocide	0.0864	1.09
	(0.59)	
Prefectures with very intense genocide	0.404***	1.498
	(3.09)	
Household characteristics		
Urban	-0.259	0.772
	(0.97)	
Wealth index score	-0.0621	0.94
	(0.53)	
Access to improved water source	-0.254	0.776
	(1.90)	
Household head is female	0.167	1.181
	(1.42)	
Maternal characteristics		
Mother knows oral rehydration	-0.186	0.83
	(1.19)	
Mother used contraception at least once	-0.204	0.816
	(1.50)	
Mother is able to read	-0.369***	0.691
	(3.37)	
Child characteristics		
Mother's age at birth	-0.118	0.888
	(1.39)	
Mother's age at birth squared	0.00211	1.002
	(1.53)	
First-born	0.468	1.596
	(1.89)	
24-35 months since previous birth (base:1-23 months)	-0.676***	0.509
	(5.07)	
36 or more since previous birth	-0.917***	0.4
	(6.16)	
Male	0.238**	1.269
	(2.3)	
Number of observations	2,889	
Wald Chi2	6226.96	Prob >chi2=0.0000

Notes: *,**,***: significant at the 10, 5 and 1 per cent respectively. *t* statistics in parenthesis. The exponentiated coefficients represent the hazard ratios from the underlying continuous time model. Standard errors are clustered at the community level. Duration dependence is accounted for by means of 12 monthly dummies which are jointly significant at 1 per cent. Regression based on weighted data.

Both intensity of exposure at the at the individual level (*Ratio of deaths among maternal siblings*) and at the prefecture level (*Prefectures with very intense genocide*) are significantly and positively associated with the hazard of dying during the first year of life. These two variables increase the hazard ratios by large amounts (88 and 50 percentage points, respectively). This result suggests that these two variables are correctly approximating exposure, since they are probably capturing the cases who have died because of the conflict.

As for the other control variables, mother's literacy, for example, reduces the hazard rate by 30 percentage points. Timing of birth has a large effect: the longer the time elapsed from previous births, the lower the risk of mortality (50 percentage points 24-35 months since previous birth and 40 percentage points more than 35 months since previous birth). [17]

[17] We did not find any relevant role for frailty. The reported standard errors are clustered at the community level. We also grouped standard errors at the province level, but this did not produce any sizeable change.

VI Child Mortality: Results

Table 3 presents the descriptive statistics of the child mortality model.

Table 3 *Descriptive Statistics for Child Mortality*

Variables	Model 2 In life and *in utero* esposure		Model 3 *In utero* esposure	
	mean	s.d	mean	s.d
Genocide intensity				
Ratio of deaths among maternal siblings 1994	0.11	0.22	0.11	0.22
Prefectures with not intense genocide	0.35	0.48	0.36	0.48
Prefectures with moderate intensity	0.32	0.47	0.34	0.47
Prefectures with high intensity	0.33	0.47	0.31	0.46
Mother changed prefecture of residence	0.12	0.33	0.13	0.33
Months of exposure	2.36	1.83	1.71	1.85
Household characteristics				
Urban	0.18	0.38	0.18	0.38
Wealth index score	-0.10	0.87	-0.09	0.88
Access to improved water source	0.82	0.39	0.81	0.39
Household head is female	0.33	0.47	0.33	0.47
Maternal characteristics				
Mother knows oral rehydration	0.89	0.31	0.89	0.31
Mother used contraception at least once	0.28	0.45	0.27	0.45
Mother can read	0.59	0.49	0.59	0.49
Child characteristics				
Mother's age at birth	28.28	6.52	28.28	6.63
First-born	0.23	0.42	0.24	0.43
1-23 months since previous birth	0.25	0.43	0.23	0.42
24-35 months since previous birth	0.27	0.44	0.27	0.44
36 or more months since previous birth	0.49	0.50	0.50	0.50
Male	0.50	0.50	0.49	0.50
Number of observations	2,099		1,442	
Months of birth	04/1994-12/1995		08/1994-12/1995	

As explained, Model 2 and 3 aim at capturing the longer term impact of the genocide on the hazard of dying between the fourth and the sixtieth month of life. Table 4 shows the coefficients of the estimated models.

Table 4 *Child mortality of the survivors: Complementary Log Log regression*

	Model 2 Coefficients	Exponentiated Coefficients	Model 3 Coefficients	Exponentiated Coefficients
Exposure to genocide				
Ratio of deaths among mat. siblings in 1994	0.266	1.304	0.242	1.274
	(0.9)		(0.59)	
Months of Exposure	0.0958***	1.101	0.10**	1.106
	(2.64)		(2.43)	
HH changed pref. of res. during the genocide	-0.0419	0.959	-0.0695	0.933
	(0.21)		(0.29)	
Living in moderately exposed prefecture	-0.119	0.888	-0.0165	0.984
	(0.79)		(0.09)	
Living in higly exposed prefecture	0.09	1.094	0.272	1.312
	(0.59)		(1.42)	
Household characteristics				
Urban	-0.314	0.73	-0.0462	0.955
	(0.93)		(0.12)	
Wealth index score	-0.151	0.86	-0.279	0.757
	(0.79)		(1.07)	
Access to improved water source	-0.376**	0.687	-0.364**	0.695
	(2.57)		(2.03)	
Household head is female	0.134	1.143	-0.0991	0.906
	(0.98)		(0.57)	
Maternal characteristics				
Mother knows oral rehydration	-0.0952	0.909	-0.0279	0.972
	(0.48)		(0.11)	
Mother used contraception at least once	-0.191	0.826	-0.106	0.899
	(1.15)		(0.52)	
Mother is able to read	-0.315**	0.73	0.327**	0.721
	(2.38)		(2.02)	
Child characteristics				
Mother's age at birth	0.0266	1.027	0.0482	1.049
	(0.27)		(0.41)	
Mother's age at birth squared	-0.00106	0.999	-0.00124	0.999
	(0.64)		(0.63)	
First-born	0.357	1.429	0.69**	1.994
	(1.39)		(2.11)	
24-35 months since previous birth	-0.494***	0.61	-0.494**	0.61
	(3.02)		(2.51)	
36 or more since previous birth	-0.793***	0.453	-1.038***	0.354
	(4.07)		(4.04)	
Male	0.0391	1.04	-0.00972	0.99
	(0.31)		(0.06)	
Number of observations	2,099		1,442	
Wald Chi2	8442.31 Prob		5636.95 Prob	

Notes: *,**,***: significant at the 10, 5 and 1 per cent respectively. *t* statistics in parenthesis. The exponentiated coefficients represent the hazard ratios from the underlying continuous time model. Standard errors are clustered at the community level. Duration dependence is accounted for by means of 6 dummies (D5-D6, D7-D12, D13-D24, D25-D36, D37-D48, D49-D60) which are jointly significant at 1 per cent. Regression based on weighted data.

The genocide effect on child mortality stems from the *Number of months of exposure*: in both models, an additional month of exposure increases the hazard of dying by 10 percentage points Since average exposure is 2.36 months the average increase of the hazard rate of mortality in the sample of survivors is around 23 percentage points.

At variance with the preceding model, genocide intensity in the prefecture of residence and ratio of deaths among maternal siblings are no longer significant. In this mortality model, genocide intensity is attributed according to residence also to children who were not even conceived during the genocide. This seems to be a reasonable choice, since, the unexposed children in our sample were born right after the war, and were likely to suffer similar deprivations with respect to the exposed children who had survived. To disentangle the effects of exposure from those of genocide intensity we also introduce all interaction terms, but they are not significant.

Summarizing, the genocide is associated with an increase in child mortality of the survivors, even if exposure was only while *in utero*. This result is even more impressive if one notes that, in our setting, it is conditional on surviving at least for four months.

VII. Concluding remarks.

This paper exploits difference across birth cohorts during the Rwandan genocide to estimate the effects of civil war exposure on child mortality. Despite adults, and males in particular, were the main target of the violent conflict, we find that, considering both *in utero* and postnatal war exposure, the impact of the genocide has increased infant and child mortality by significant amounts.

Our results for infant mortality show that the differential intensity of exposure to the genocide both at the household and at the prefecture level matters, thus indicating that aid intervention should not be indiscriminate, but well targeted and context-based even during emergencies.

As far as the temporal extent of the effect of the genocide is concerned, our results show that children born during the genocide and who survived it, and even children who experienced it only *in utero*, continue to have higher mortality rates after the war. We estimate that the increase in the hazard rate of child mortality in the sample of survivors is around 10 percentage points for any additional month of exposure.

We interpret these results as evidence that what matters for child mortality rates of the survivors is the individual experience of the genocide. We reckon this to be a helpful indication for identifying the fragile generations of children to target with policy actions aimed at improving child health after a war.

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