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F. Ciani and G.C. Giannelli

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Dipartimento di Scienze Economiche, Università degli Studi di Firenze
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Surviving the genocide: the impact of the Rwandan genocide on child mortality

Federico Ciani* (University of Florence)

Gianna C. Giannelli** (University of Florence and IZA)

December 2011

Abstract

The aim of this paper is to investigate the consequences of the Rwandan genocide on infant and child mortality. Between April and July 1994 Rwanda experienced a tremendous wave of inter-ethnic violence that caused at least 500.000 deaths. We use the Rwanda DHS 2000 survey to test if exposure to the genocide has induced an increase in infant and child mortality. Considering both direct exposure to the conflict and exposure while in *utero*, we estimate several specifications of discrete-time survival models with piecewise constant baseline hazards. Our results show that the conflict increases significantly infant mortality, and that, several years after the end of the war, this effect is still there to undermine the survival of children who were exposed to it.

JEL Classification: I20, J13, O12, Z13

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

* Federico Ciani, University of Florence, Department of Economics, E-mail: federico.ciani@unifi.it

** Gianna Claudia Giannelli (corresponding author), University of Florence, Department of Economics, E-mail: giannelli@unifi.it

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1 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” (UN, 1996).

This dramatic consideration, warns nations about the disruptive effects of wars on the generations of children hit by a conflict. These effects, involving children’s health and human capital accumulation, may seriously compromise the recovery of a country, long after the end of the conflict.

Economists are aware that negative shocks affecting children early in life may lead to lower adult height, less cognitive achievement and human capital accumulation, lower productivity and wages, and higher mortality, particularly in low income countries (Strauss and Thomas 2008). In particular, among different types of shocks, the impact of wars has been analysed in relation to shocks at birth and adult outcomes several years after the shock. Akresh et al. (2009 and 2011), for example, find that children exposed to the war have on average 0.515 standard deviations lower height-for-age z-scores than non-exposed in Burundi, and 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. As far as human capital accumulation is concerned, evidence shows the negative impact of armed conflicts on children’s schooling . For example, Poirier (2011) measures the impact of war on education on 43 African countries from 1950 to 2010 and finds that especially civil wars have negative effects on school enrolment rates in the countries studied. Akresh and de Walque (2008) find a strong negative impact of Rwanda’s genocide on children’s schooling, with exposed children completing one-half year less education, which amounts to an 18.3 per cent decline in school completion. Shemyakina (2011) finds that exposure to violent conflict had a large and statistically significant negative effect on the enrolment of girls (not of boys) in Tajikistan.

Surprisingly enough, in this growing body of literature, little attention has been paid so far to the most important problem of children’s survival after a conflict.

In this paper we address this issue, analyzing the impact of the 1994 Rwandan genocide on Rwandan children’s survival. In particular, we investigate the levels and the determinants of infant and child mortality in the cohorts of children who have been exposed to the genocide, as compared to children who were not exposed. Our objective is mainly to ascertain whether the impact of a war on child mortality (which is evidently expected to be there) produces lasting effects after the end of the war. In other words, our research question is the following: even if a population recovers its initial dimension after a conflict, does the war leave behind a “permanently fragile” generation of children who have been exposed to it?

A positive answer to this question has relevant policy implications, since intervention, in such case, should not be limited to face the war and immediate post-war phase, but should plan middle-term, targeted actions to support the fragile generation.

We develop a survival analysis to investigate the consequences of the 1994 Rwandan genocide on Rwandan child and infant mortality using the 2000 Rwandan Demographic and Health Survey (RDHS), coupled with information on the genocide drawn from the data collected on the genocide by Genodynamics.[1] From the methodological point of view, the Rwandan case is particularly suitable for such analysis for two reasons. Firstly, 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. Secondly, the conflict was extremely concentrated in time (four months), so that the macroeconomic background is likely to have remained the same for both the exposed and the unexposed children , thus reducing the problem of controlling for changing economic conditions.

We focus both on infant mortality (i.e. child mortality in the first year of life) and, in order to disentangle the long-run effects of the war, on the child mortality (i.e. child mortality in the first five year of life) of a sample of children who survived after the war. Moreover, a specific analysis conducted only on the exposed children allows introducing some genocide variables to measure the intensity of the conflict and its consequences on child mortality .

Considering both direct exposure to the conflict and exposure while in *utero* - as it is proved that psychophysical conditions during gestation have a significant impact over unborn children's health - we estimate several specifications of discrete-time survival models with piecewise constant baseline hazards.

Our results indeed show that the conflict increases significantly infant mortality and that several years after the end of the war, this effect is still there.

The paper is structured as follows. Section 2 introduces briefly the Rwandan genocide and Rwandan child and infant mortality. Section 3 describes the data, the model specification and the sample selection. Section 4 presents the results of the survival analysis and Section 5 concludes discussing some implications for policy.

2 The Rwandan genocide and child mortality

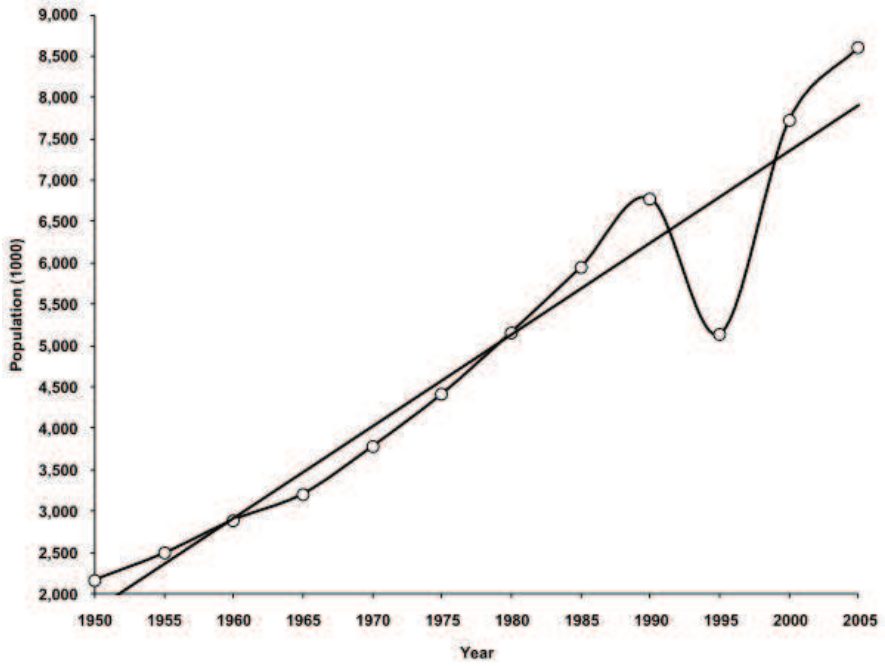
The African Great Lakes region has been disrupted by civil and inter-state wars, genocides and coups d'état since the mid nineties. Only nowadays it is possible to glimpse a feasible path of stabilization of the region even if armed conflicts are still standing (for example in the Kivu region in the 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 (during the period ranging from April to July 1994) between 500.000 and 800.000[2] Tutsi and

[1] See Genodynamics, at www.genodynamics.com

[2] These estimates are those provided by the OAU (1999) in the report "Rwanda: the Preventable Genocide".

moderate Hutu[3] were killed by the Rwandan army, by the police and by the members of the Interhamwe militia.[4] According to the estimates reported by Gardemann (2002), the total number of deaths, including those indirectly due to the genocide (for example deaths occurred among displaced people), reached one million. 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, doesn't exclude the existence of long term negative consequences of the genocide for the Rwandan population.

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. Carbone (2000) 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 sharp fall of the price of coffee (Chossudowsky [2003]).

At any rate, as far as mortality is concerned, the 1994 Rwandan events can be surely classified as a "complex emergency" that is, a situation "in which mortality among the civilian population substantially increases above the population baseline either as a result of the direct effects of war or indirectly through increased prevalence of malnutrition and/or transmission of communicable diseases, particularly if the latter results from deliberate political and military policies and strategies" (Salama et al., 2004, p. 1801). Complex humanitarian emergencies are not a simple transitional deviation from the usual development path: the

[3] Hutus that were not supporters of the "Hutu power" ideology.
[4] Detailed data and information concerning Rwandan genocide are available on the website www.genodynamics.com.

sudden loss of infrastructure, social cohesion, wealth etc., structurally requires external aid to be immediately recovered.[5]

One of the most relevant aspects of complex emergencies is their impact on child welfare (Moss et al. [2005]). In fact, during each phase of the emergency, children's specific needs and vulnerability factors need to be addressed. As an example, one can think of the problem of meeting the daily nutritional requirements of breast-fed children. Breast-fed children depend on a peculiar and not easily replaceable food item, breast milk whose supply is heavily affected by the mothers' psychophysical conditions. Generally, children are more vulnerable than adults to consequences of micro nutrients deficiencies and, consequently, are more likely to suffer permanent consequences due to transitory deprivation. Similar considerations can be made about the exposure to specific diseases (e.g. diarrhoea or respiratory infections).

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[6] adult males, who were richer and more educated than the average of the population (De Walque et al. [2009]). So, as long as children are not an explicit target of the violence committed during the genocide, we are going to analyze not only the impact of direct violence but also the indirect consequences of the events occurred between April and July 1994.

3 Data, model specification, sample selection and variables

The main aim of this paper is to investigate the impact of the 1994 Rwandan genocide on Rwandan children's mortality. Following the current definitions of child mortality (see for example 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).

The Rwandan Demographic and Health Survey (RDHS) for 2000[7] records each woman's "birth history" collecting recall information on each birth in her lifetime up to the time of the interview.[8] With these data, we reconstruct the birth histories of 27602 children belonging to 6539 households, including those of children who died before the date of the interview. Also, 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.

[5] Complex humanitarian emergencies have immediate, medium term and long term consequences. Their progress can be generally divided into three phases: (i) early\acute emergency phase, 0-1 month; (ii) late\recovery phase, 1-6 months; (iii) rehabilitation\development phase: 6 and more months (Health Frontiers [2005]).

[6] 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.

[7] RDHS 2000 is the first available survey after the genocide.

[8] The interviews took place between June 26 and November 30, 2000.

Information on children's month and year of birth - and also 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.[9] Measuring time in monthly intervals *j* indexed by the positive integers, we choose a complementary log log functional form[10] 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 year than in the subsequent years). Under these assumptions, γ_j in (1) can be expressed 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.

Another problem is represented by frailty. DHS surveys have a stratified structure: individuals are nested into households, households into communities and communities into regions. Ignoring this stratification leads to underestimating the standard errors and, consequently, to overestimating the significance of coefficients. In order to avoid this shortcoming, we choose to compute clustered standard errors, allowing for intra-group correlation.[11]

[9] To estimate the model we need to transform the data in person-periods, that means having for each child in the data matrix as many rows as the months at risk.

[10] The *cloglog* model is the discrete time representation of a proportional hazards model (see Jenkins [2004, 1997, 1995]). Another functional form is the logistic, but it did not yield significantly different results.

[11] Another strategy would be to adopt a multi-level structure for the model. This choice would be, however, computationally heavy.

We estimate three model specifications. In the first two models we analyse infant mortality, where our dependent variables is child's survival up to the first year of life and we estimate the probability of dying before the first birthday. In the third model we take a different perspective and focus on the consequences for survival up to five years of having experienced the genocide in the first months of life and having survived until the end of it. Given the duration structure of our data, each model must be estimated on the appropriate sub-sample of the population.

3.1 Sample of model 1: infant mortality

The first model tests whether the genocide had an effective impact on infant mortality. For this scope, we select the cohorts of children born between May 1993 and July 1994, namely, children who have been exposed to the genocide during their first year of life. In order to test whether the genocide impacted children exposed to it while *in utero*, we include in the sample children born between August 1994 and April 1995.[12] In fact, it seems sensible to suppose that pregnant mother's psychophysical conditions (surely challenged by the genocide and its consequences) have a significant impact over soon-to-be-born child's health.[13] According to this sample selection rule, the sub-sample of "exposed" children is composed by 2492 children. We add to this sub-sample two control groups. These control groups are chosen according to the same temporal definition: the first group includes children born during the year preceding the genocide, namely, between May 1991 and April 1993. The second control group includes the cohort of children born during the year following the genocide, namely, between May 1995 and April 1997. These two sub-samples are respectively formed by 2228 and 2831 children. The total number of survival spells expressed in monthly data in the first model is therefore 83613. 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 had the opportunity to survive one year.

In Table 1, the observed children are distributed according to their group and to their month of birth. For each monthly cohort, the percentage of children who died during their first year of life is reported. The last row reports the total number of children and the total percentage of deaths for each group. It is easy to notice that the percentage of deaths among the exposed children is the highest while the percentage for the post-genocide control group is the lowest. This evidence seems to suggest that the genocide has induced a temporary deviation from a virtuous trend improvement of infant survival conditions.

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

[13] Outcomes and stresses suffered while in *utero* have been investigated by Barker (1998). The seminal work by Stein et al. (1975) finds that the cohort in *utero* during the 1944-45 Dutch famine exhibited a range of negative health outcomes as adults.

Table 1.
Infant mortality by monthly birth cohort (exposed children and control groups)

Pre-genocide control group			Exposed group			Post-genocide control group		
month of birth	No. of obs.	% deaths during the 1st year of life	month of birth	No. of obs.	% deaths during the 1st year of life	month of birth	No. of obs.	% deaths during the 1st year of life
may-91	61	14.75	may-93	112	15.18	may-95	104	9.62
jun-91	85	12.94	jun-93	100	9.00	jun-95	107	7.48
jul-91	92	4.35	jul-93	130	13.08	jul-95	131	8.40
aug-91	84	9.52	aug-93	103	13.59	aug-95	137	5.84
sep-91	79	6.33	sep-93	85	16.47	sep-95	125	9.60
oct-91	65	6.15	oct-93	88	14.77	oct-95	101	11.88
nov-91	50	8.00	nov-93	62	22.58	nov-95	96	10.42
dec-91	57	19.30	dec-93	81	11.11	dec-95	90	10.00
jan-92	111	8.11	jan-94	103	15.53	jan-96	152	4.61
feb-92	96	17.71	feb-94	133	12.78	feb-96	116	12.93
mar-92	91	9.89	mar-94	121	10.74	mar-96	105	8.57
apr-92	138	10.87	apr-94	195	17.44	apr-96	125	11.20
may-92	109	12.84	may-94	121	12.40	may-96	136	8.09
jun-92	118	11.02	jun-94	142	21.83	jun-96	154	12.99
jul-92	117	8.55	jul-94	160	11.88	jul-96	143	11.89
aug-92	142	12.68	aug-94	131	13.74	aug-96	119	7.56
sep-92	78	16.67	sep-94	114	14.91	sep-96	109	4.59
oct-92	80	8.75	oct-94	97	12.37	oct-96	106	16.98
nov-92	54	22.22	nov-94	60	25.00	nov-96	112	9.82
dec-92	73	6.85	dec-94	77	20.78	dec-96	95	10.53
jan-93	98	11.22	jan-95	72	6.94	jan-97	103	6.80
feb-93	114	14.91	feb-95	63	12.70	feb-97	113	15.04
mar-93	112	12.50	mar-95	64	12.50	mar-97	108	2.78
apr-93	124	13.71	apr-95	78	14.10	apr-97	144	14.58
Total	2228	11.54	Total	2492	14.53	Total	2831	9.68

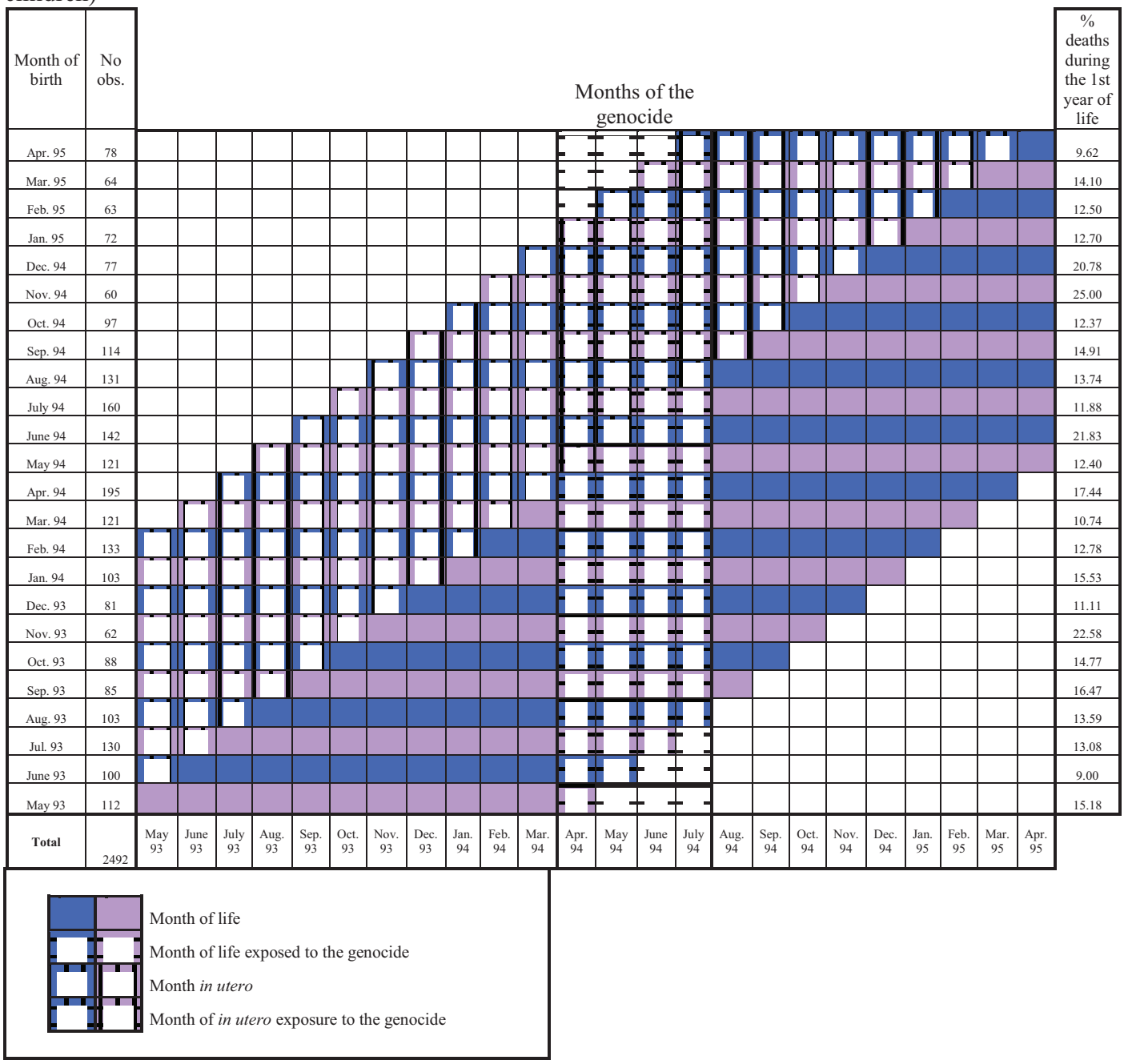
Source: our elaboration on RDHS 2000

3.2 Sample of model 2 : infant mortality of children exposed to genocide

In the second model we try to deepen some aspects of the exposure to violence. Consequently the sample is restricted to the 2492 children exposed (both directly and *in utero*) to the genocide.

Table 2 reports more detailed information concerning the generations of children exposed to the genocide.

Table 2. Structure of exposure to the genocide and infant mortality by monthly birth cohort (only exposed children)



Source: our elaboration on RDHS 2000

The table shows the structure of exposure to the genocide for each monthly cohort of children distinguishing between direct and *in utero* exposure. For example, the 142 children born in June 1994 were directly exposed to the genocide for two months and *in utero* for other two months.

3.3 Sample of model 3: child mortality

The third model aims at investigating the longer term consequences of the genocide. In this case, our dependent variable becomes the probability of dying between the fifth and the sixtieth month of life. As for

the sample for model 1, also in this case the sample includes a group of exposed children and a control group. We select children born after March 1994, who survived the first four months of life.[14] Moreover, we drop children who were not potentially able to turn five before the month of interview in 2000 – whether alive or not at the time of the interview. This sample consists of 1772 individuals and 97306 survival spells. In this model, we control for the length of the direct and *in utero* exposure to the genocide, which ranges from 0 to 4 months. Table 3 summarizes the distribution of the sample according to the length and the kind of the exposure to the genocide.

Table 3. Number of months of exposure to the genocide in the child mortality model (exposed and unexposed children)

No. of months of exposure	Direct exposure		*In utero* exposure		Total exposure	
	No. of children	%	No. of obs.	%	No. of obs.	%
0	1225	69.13	953	53.78	669	37.75
1	144	8.13	119	6.72	0	0.00
2	119	6.72	144	8.13	0	0.00
3	111	6.26	176	9.93	550	31.04
4	173	9.76	380	21.44	553	31.21

Source: our elaboration on RDHS 2000

3.4 Genocide variables and other controls.

As the main goal of this paper is to investigate the impact of the 1994 Rwandan genocide on Rwandan children's mortality, an appropriate set of variables describing (or at least approximating) the exposure to and the dimension of the genocide is needed.

In order to control for the exposure, we introduce in Model 1 two dummies:"pre-genocide control group" and "post-genocide control group" equal to one respectively if the child is born between June 1991-April 1993 and during the period May 1994-March 1995. Consequently, the generation of children born between May 1993-March 1995 is considered as the baseline category. Note that in this model with exposed and unexposed children we approximate exposure in no other way than with these dummy variables, so that it would be more precise to talk about "potential exposure" to the genocide. Moreover, some of the "potentially" exposed children did not survive till the beginning of the genocide.

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). Among them, a urban\rural dummy is included in the three estimated models. In Model 1 we also include a set of dummies for Rwandan prefectures (with Kigali as reference categories). In the other two models these dummies are not included, as we use other prefecture-level dummies to describe the genocide, and the two sets of dummies might be collinear.

[14] We therefore exclude right censored survival spells, since they would only be observed for unexposed children.

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 diarrhoea and other gastro-enteric diseases (Victora et al. [2000] and UNICEF [2008]). So we include a dummy for mother's knowledge of oral rehydration therapy.

As one of the main sources of contamination is water, the presence in the household of an improved source of water is an important factor to improve child health. 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. A dummy variable indicating whether a household has access to an improved water source is included in the three estimated models.

In order to investigate the relationship between women's empowerment and child mortality a dummy variable equal to one if child's mother has ever used a modern contraceptive method is therefore included in the three estimated models. Therefore, contraception is introduced in the model as a proxy of women's autonomy and emancipation (Eswaran [2002]). The association between mothers' education and children's health is one of the most investigated and verified relationships (see, for example, the seminal work by Mosley and Chen [1982] and Adetunji [1995] or Cochrane et al. [1982]). 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.

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][15]). Mother's age at birth is included in the three models as a continuous variable with a quadratic specification.

Birth spacing is very important for mothers' and children's health. Close births tend to put mothers' physical condition under pressure and children born in this condition tend to be more fragile (Winikoff [1983]). So a categorical variable concerning birth interval (with the class 1-23 months as reference category) has been introduced into the three models.

Birth order might be a relevant factor as concerns children's survival. In fact, the first birth and high order births usually are more dangerous than second, third and fourth births (Miller et al. [1992]).

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

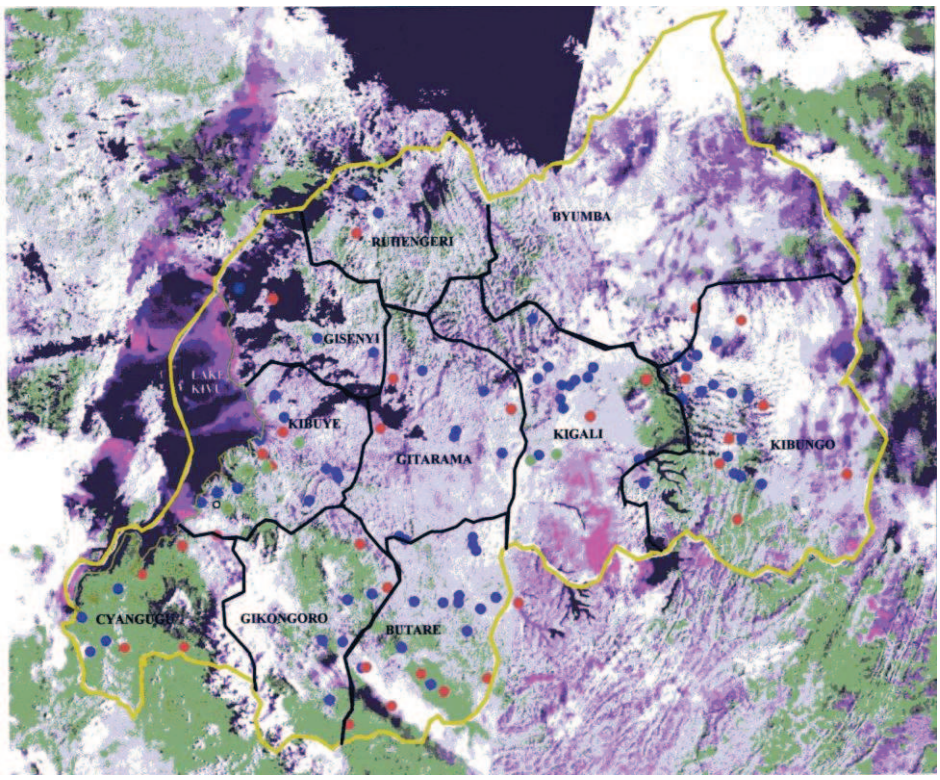
[15] The authors find, for the US, that older maternal age has an adverse effect on a child's educational outcome regardless of whether other factors are controlled for or not. Instead, the association of young maternal age and long-term morbidity is not significant when controlling for other factors.

The effect of wealth in the three models 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.

Model 2 focuses on the exposed children and includes other variables approximating the intensity of exposure with information related to the genocide available at the level of prefectures.

In the attempt of capturing husband's violent death, we introduce a dummy equal to one when the mother is widowed. This is a poor approximation, as we don't know anything about the causes of the husband's death. RDHS 2000 also includes a special section where information about the interviewed women's siblings is collected. We use this information to calculate a variable indicating the ratio between the number of siblings who died in 1994 and the total number of siblings . This variable might be a quite good proxy of the level of violence or, more in general, of a stressful situation faced by the household during the genocide (see De Walque and Verwimp, 2009). The model includes also prefecture-level information drawn from the Genodynamics[16] database (Davenport and Stam [2008]). A first measure is a dummy equal to if the children is living in either the prefectures of Butare, or Kigali or Kibongo, namely, the prefectures characterized by the highest number of victims according to the Genodynamics database (we call this variable "most exposed prefectures"). A second measure is the number of days in which violent episodes were recorded in the prefecture. Finally, we have included a variable indicating the number of mass graves sites detected and signalled by the Rwandan Genocide Project (2007; see Figure 2).

Figure 2. Mass Graves Sites and Memorials in Rwanda



[16] Data are downloadable from the website www.genodynamics.com

Purple and red dots represents respectively mass graves and memorials
Source: Rwandan Genocide Project (2007)

In Model 3 a further variable has been added to approximate the individual intensity of the exposure to the genocide. It is a numeric 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*. We have used this variable only in this model, since we deal with mortality between 4 and 60 months. In the other two models, measuring the impact in months would have a misleading consequence. In fact, if a child has been exposed for four months to the genocide, it means that it has survived at list for four months after their birth. As the first months of life are physiologically characterized by the highest probability of death, the variable "months of exposure to the genocide" would be positively correlated with child survival. The analysis of Model 3 focuses on the survival after the fourth month of life and, consequently, this shortcoming is avoided.

Table 4 reports the descriptive statistics for subsamples 1 and 2. Notice that the statistics are computed for the whole population and for each group (children exposed to the genocide, pre- and post-genocide control groups).

Table 4. Descriptive Statistics for Model 1 and Model 2

Variables	Total population (model 1)		Pre-genocide		Exposed (model 1,2)		Post-genocide	
	mean	s.d	mean	s.d	mean	s.d	mean	s.d
State fixed effects								
Kigali	0.228	0.356	0.204	0.384	0.222	0.380	0.252	0.328
Butare	0.093	0.291	0.104	0.305	0.082	0.274	0.095	0.294
Cyangugu	0.097	0.296	0.092	0.289	0.103	0.304	0.095	0.294
Gikongoro	0.086	0.280	0.089	0.285	0.079	0.269	0.089	0.285
Gisenyi	0.081	0.273	0.079	0.269	0.087	0.283	0.078	0.268
Gita rama	0.088	0.284	0.093	0.291	0.084	0.278	0.088	0.284
Kibungo	0.102	0.302	0.105	0.307	0.101	0.302	0.100	0.300
Kibuye	0.108	0.311	0.112	0.316	0.112	0.316	0.102	0.303
Ruhengeri	0.116	0.321	0.122	0.327	0.130	0.336	0.100	0.300
Household characteristics								
Urban	0.203	0.402	0.185	0.388	0.193	0.395	0.225	0.418
Wealth index score	-0.037	0.954	-0.084	0.927	-0.604	0.923	0.020	0.923
Access to improved water source	0.827	0.378	0.827	0.379	0.820	0.384	0.835	0.372
Household head is female	0.346	0.476	0.416	0.493	0.353	0.479	0.285	0.452
Maternal characteristics								
Mother knows oral *rehydration*	0.881	0.324	0.878	0.327	0.878	0.328	0.886	0.318
Mother used contraception at least once	0.277	0.448	0.264	0.441	0.279	0.449	0.286	0.452
Mother can read	0.591	0.492	0.532	0.499	0.580	0.494	0.647	0.478
Child characteristics								
Mother's age at birth	28.527	6.547	28.584	6.317	0.289	6.510	28.195	6.740
First-born	0.220	0.415	0.201	0.401	0.200	0.400	0.254	0.435
1-23 months since previous birth	0.252	0.430	0.273	0.458	0.268	0.460	0.223	0.439
24-35 months since previous birth	0.262	0.440	0.260	0.439	0.258	0.438	0.266	0.442
36 or more since previous birth	0.486	0.500	0.467	0.499	0.474	0.499	0.511	0.500
Birth order	3.837	2.514	3.885	2.442	3.999	2.536	3.657	2.539
Male	0.496	0.500	0.500	0.500	0.493	0.500	0.500	0.500
Exposure to genocide								
Pre-genocide control group	0.295	0.456	1.000	0.000	0.000	0.000	0.000	0.000
Exposed group	0.330	0.465	0.000	0.000	1.000	0.000	0.000	0.000
Post-genocide control group	0.375	0.484	0.000	0.000	0.000	0.000	1.000	0.000
Genocide intensity								
Mother is widow	0.0045	0.06696	0.004	0.634	0.004	0.066	0.005	0.070
No. of days of violence in the prefecture	29.005	34.987	29.373	35.531	29.226	35.593	28.521	34.011
No. of mass graves in the prefecture	8.470	4.45125	8.442	4.471	8.280	4.497	8.658	4.389
Most exposed prefectures	0.305	0.46043	0.313	0.464	0.293	0.455	0.310	0.462
Ratio no. of dead to total siblings	0.089	0.19243	0.093	0.198	0.086	0.187	0.088	0.192

Source: our elaboration on RDHS 2000

Table 5 reports the descriptive statistics for Model 3.

Table 5. Descriptive Statistics for Model 3

Variables	mean	s.d
Household characteristics		
Urban	0.196	0.397
Wealth index score	-0.052	0.925
Access to improved water source	0.840	0.367
Household head is female	0.346	0.476
Maternal characteristics		
Mother knows oral *rehydration*	0.895	0.307
Mother used contraception at least once	0.284	0.451
Mother can read	0.608	0.488
Child characteristics		
Mother's age at birth	28.336	6.522
First-born	0.227	0.258
1-23 months since previous birth	0.249	0.432
24-35 months since previous birth	0.258	0.438
36 or more months since previous birth	0.493	0.500
Birth order	3.795	2.529
Male	0.494	0.500
Genocide intensity		
Months of direct exposition to genocide	0.794	0.925
Months of in utero exposition	1.802	1.806
Most exposed prefectures	0.279	0.449
Ratio of no. of dead to total siblings prefectures	0.087	0.188

Source: our elaboration on RDHS 2000

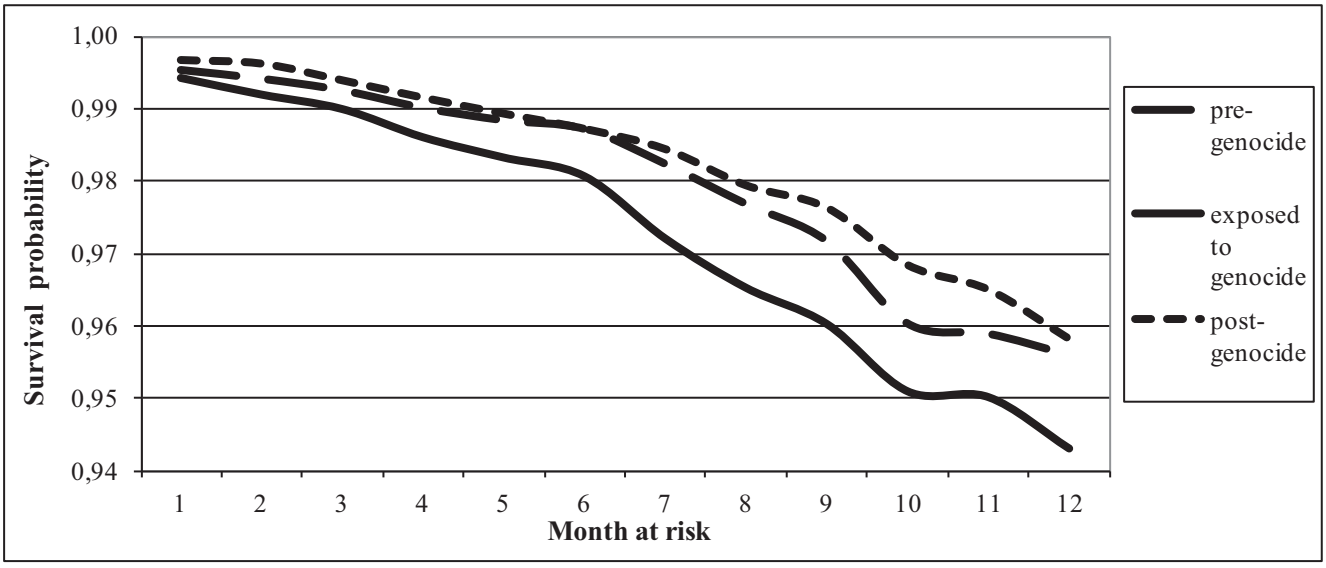
4 Analysis

Tables 6-9 report the coefficients and the standard errors of the discrete time survival analysis for the three models.

4.1 Model 1: infant mortality

The main aim of this model is to test whether exposure to the genocide lowers the survival probability of children during their first year of life. This issue can be described non-parametrically by means of the survival curves (see Figure 3).

Figure 3. Survival curves for exposed and not exposed children



Source: our elaboration on RDHS 2000

The survival curve of children belonging to the generation exposed to the genocide (described in Table 2) lies under the survival curves of the control groups (described in Table 1). This means that, on average, the exposed children have worse survival conditions than control groups. The significance of the difference between the survival curves can be tested through a log-rank test for the equality of survival functions. The test clearly suggests the rejection of the null hypothesis of equality between the curves (see Table 6).

Table 6. Log-rank test for the equality of survival functions

Group	Events observed	Events expected	chi2(2)	Pr>chi2
pre-genocide	1048	1099.6		
exposed to genocide	1440	1193.05	80.74	0.0000
post-genocide	1229	1424.35		

Source: our elaboration on RDHS 2000

The results of the parametric *cloglog* duration analysis of infant mortality are presented in Table 7. The estimated coefficients measure the hazard of dying during the first year of life. There is not any significant difference in survival probabilities between children born in rural and urban areas. As concerns the prefecture fixed effects, the prefectures of Cyangugu, Ruhengeri and Kibuye are characterized by more favourable survival conditions than in Kigali. There is a significant negative correlation between the probability of dying during the first year of life and household wealth and a negative association with mother's ability to read. We also notice a negative significant effect of contraception use and oral rehydration knowledge on the hazard. Sex of the child and of the household head, and access to improved water sources are not (or only marginally) significant variables. Access to water might turn out be not significant as the effect may be caught by wealth. Duration dependence is overall significant.[17]

Turning to our major scope, potential exposure to the genocide has a significant and negative association with survival, this meaning that, on the whole, even after controlling for other variables, the generation of children who experienced at least one month of genocide during their first year of life or *in utero*, had a significantly higher hazard of dying than children belonging to the control groups.[18] In terms of odd-ratios, the pre-genocide control group had 25 and the post-genocide control group had 32 percentages point less probability to die than the exposed group, respectively. This is a quite relevant effect, compared, for

[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.

[18] An alternative specification of Model 1 with interaction terms between the dummies concerning exposure to the genocide and the other variables was estimated. This specification was potentially interesting, since interaction terms could capture the differences in the role of control variables between the exposed and the unexposed. However, these interaction terms have turned out to be not significant.

example, to mother's literacy, which improves the probability to survive by a similar amount (0.26 percentage points).

Table 7. Results for Model 1: infant mortality (exposed and unexposed children)

Variables	Coefficient		Robust S. E.
State fixed effects			
Kigali	-		-
Butare	0.209		0.161
Cyangugu	-0.398	(*)	0.205
Gikongoro	-0.123		0.169
Gisenyi	*-0.267*		*0.184*
Gita rama	0.125		0.187
Kibungo	0.115		0.164
Kibuye	-0.392	(**)	0.169
Ruhengeri	*-0.300*	(*)	*0.164*
Household characteristics			
Rural	-		-
Urban	0.054		0.202
Wealth index score	-0.167	(**)	0.074
No access to improved water source	-		-
Access to improved water source	*-0.122*		*0.099*
Household head is male	-		-
Household head is female	0.029		0.083
Maternal characteristics			
Mother does not know oral rehydration	-		-
Mother knows oral rehydration	-0.227	(**)	0.105
Mother never used contraception	-		-
Mother used contraception at least once	-0.161	(*)	0.092
Mother is not able to read	-		-
Mother is able to read	-0.294	(***)	0.081
Child characteristics			
Mother's age at birth	-0.160	(***)	0.051
Mother's age at birth squared	0.002	(***)	0.001
First-born	0.691	(***)	0.149
1-23 months since previous birth	-		-
24-35 months since previous birth	*-0.548*	(***)	*0.091*
36 or more since previous birth	-1.108	(***)	0.119
Birth order	*0.057*	(*)	*0.030*
Female	-		-
Male	*0.118*		*0.073*
Duration dependence			
D1 (i.e. first month of life)	0.296	(**)	0.792
D2	*-1.618*	(**)	*0.819*
D3	-1.571	(**)	0.807
D4	-1.490	(**)	0.793
D5	*-1.964*	(***)	*0.801*
D6	-2.237	(***)	0.812
D7	-1.417	(**)	0.799
D8	-1.454	(**)	0.805
D9	-1.849	(***)	0.790
D10	-1.169	(**)	0.798
D11	-2.808	(***)	0.833
D12	-2.518	(***)	0.821
Exposure to genocide			
Pre-genocide control group	*-0.284*	(***)	*0.084*
Exposed group	-		-
Post-genocide control group	-0.392	(***)	0.084

Source: our elaboration on RDHS 2000.
Standard errors are clustered at the community level
*,**,***: significant at the 10, 5 and 1 per cent respectively

4.2 Model 2: Infant mortality of the exposed children

Model 2 focuses on children exposed to the genocide and includes the variables approximating the effect of exposure to violence both at a prefecture and at an individual/household level Table 8 shows that the significance and the direction of the association between the control covariates and the hazard of dying in

the first year of life is not drastically different from the other model. The model with frailty has been estimated but unobserved heterogeneity was not significant.

As concerns the genocide, both intensity of exposure at the prefecture level (number of mass graves in the prefecture) and at the individual level (ratio between mother's dead siblings and total siblings) are significantly and positively associated with the hazard of dying during the first year of life. The latter, increases the probability by an enormous amount, nearly 90 percentage points, this meaning that with this variable we are probably capturing the cases who have died during the conflict. The other variables are not significant as they are probably poorly measured (mother is widow) or because the whole effect is caught by the other variables.

Table 8. Results for Model 2: infant mortality (exposed children)

Variables	Coefficient		Robust S. E.
Household characteristics			
Rural	-		-
Urban	-0.164-		-
Wealth index score	-0.205	(*)	0.264
No access to improved water source	-		-
Access to improved water source	-0.161		0.158
Household head is male	-		-
Household head is female	0.110		0.116
Maternal characteristics			
Mother does not know oral rehydration	-		-
Mother knows oral rehydration	-0.161		0.158
Mother never used contraception	-		-
Mother used contraception at least once	-0.203		0.141
Mother is not able to read	-		-
Mother is able to read	-0.249	(**)	0.109
Child characteristics			
Mother's age at birth	-0.129		0.083
Mother's age at birth squared	0.002	(*)	0.001
First-born	0.674	(***)	0.251
1-23 months since previous birth	-		-
24-35 months since previous birth	-0.525	(***)	0.150
36 or more since previous birth	-1.024	(***)	0.163
Birth order	0.029		0.041
Female	-		-
Male	0.188	(*)	0.113
Duration dependence			
D1 (i.e. first month of life)	-0.967		1.221
D2	-2.579	(**)	1.239
D3	-3.006	(**)	1.271
D4	-2.625	(**)	1.243
D5	-3.163	(**)	1.276
D6	-3.377	(***)	1.255
D7	-2.351	(*)	1.268
D8	-2.740	(**)	1.250
D9	-3.110	(**)	1.224
D10	-2.626	(**)	1.230
D11	-5.103	(***)	1.413
D12	-3.714	(***)	1.216
Genocide intensity			
Mother is married, divorced or never married	-		-
Mother is widow	0.299		0.722
No. of days of violence in the prefecture	-0.003		0.002
No. of mass graves in the prefecture	0.068	**(**)**	0.029
Most exposed prefecture	-0.208		0.253
Ratio no. of dead to total siblings	0.591	**(**)**	0.247

Source: our elaboration on RDHS 2000
Standard errors are clustered at the community level
*,**,***: significant at the 10, 5 and 1 per cent respectively

4.3 Model 3: child mortality

Model 3 aims at describing a longer term impact of the genocide on the hazard of dying between the fourth and the sixtieth month of life. Table 9 shows that here too, the impact of the control covariates is in line with the expectations and very similar to the other two estimated models. As for wealth, the coefficient is again negative and significant, since here the negative effect is not outweighed by the possible positive effect on the exposed children. The baseline hazard declines with age of children, this meaning that mortality rates are lower for older children. The decline tends to slow down as children become older. As concerns the variables approximating the effect of the genocide, here it is possible to include a variable describing the individual exposure to the genocide expressed in months (ranging from 1 to 4). Months of exposure to the genocide are negatively associated with survival probability: one month more of exposure increases the hazard of dying by 7 percentage points (the odds ratio is 1.07) So, even excluding the first months of life from the analysis, there is evidence of an enduring impact of the genocide on children survival conditions. The impact on survival probability during the first months of life may be partly due to direct violence and partly to the immediate consequence of the genocide (displacement etc.). If the impact is still there until the fifth year of life it means that the genocide creates a fragile generation of children whose survival probability is lower than that of other generations. Measures of the genocide intensity at the prefecture level (number of mass graves and prefecture geographic exposure) are significant too.[19]

[19] Standard errors are clustered at the community level. The role of unobserved heterogeneity in the model with frailty was not significant.

Table 9. Results for Model 3: child mortality (exposed and unexposed children)

Variables	Coefficient		Robust S. E.
Rural			
Urban	-0.1309866		0.251351
Wealth index score	-0.410101	(***)	0.156872
No access to improved water source	-		-
Access to improved water source	-0.4551817	(**)	0.159146
Household head is male	-		-
Household head is female			
Maternal characteristics			
Mother does not know oral rehydration	-		-
Mother knows oral rehydration	0.1157935		0.209352
Mother never used contraception	-		-
Mother used contraception at least once	-0.2074475		0.170944
Mother is not able to read	-		-
Mother can read	-0.301176	(**)	0.142102
Child characteristics			
Mother's age at birth	-0.2904421	(***)	0.040806
Mother's age at birth squared	0.0045622	(***)	0.000783
First-born	-0.128		0.244
1-23 months since previous birth	-		-
24-35 months since previous birth	-0.5117111	(***)	0.17626
36 or more since previous birth	-0.7493123	(**)	0.200577
Birth order	-0.0605009		0.052235
Female	-		-
Male	-0.0008901		0.132081
Duration dependence			
D5 to D12	0.4009843	(*)	0.246991
D13to D36	-0.4407482	(**)	0.234237
D37 to D48	-1.052445	(**)	0.288635
D49 to D60	-1.247458	(**)	0.304106
Genocide intensity			
Months of exposure to the genocide	0.082245	(**)	0.037248
Most exposed prefectures	0.757	(**)	0.326
Ratio of no. of dead to total siblings	0.4717158	(**)	0.343396

Source: our elaboration on RDHS 2000
Standard errors are clustered at the community level
*,**,***: significant at the 10, 5 and 1 per cent respectively

5 Conclusions

Surely, the events occurred in Rwanda in 1994 are an *unicum* in the human history. Therefore, it is necessary to be careful in drawing general lessons from the analysis of these events. In fact, complex emergencies are often peculiar events as they often find their roots in the history, in the characteristics, in the political situation of a country. Despite that, as there is a huge need of refining the ways of policy intervention, any attempt to learn something from such events seems reasonable and justified.

A first lesson that can be learnt from the Rwandan experience is that the consequences of a complex emergency are likely to hit groups that are not the most exposed during the most critical stage of the emergency. In fact we have seen that, despite adults (and males in particular) were the main target of the violent conflict, the impact of the genocide on Rwandan children was sharp and devoid of sizeable differences between males and females. Moreover, our results clearly show that the intensity of exposure to the genocide both at the individual and at the prefecture level matters, this meaning that intervention should not be indiscriminate. We have included *in utero* exposure, and we have seen that it is significant. This result suggests that pregnant mothers must be considered as a very vulnerable group, not only as concerns their own health, but also as concerns their soon-to-be-born children's health.

Another important aspect concerns the temporal extent of the impact of the genocide. Our result show that children born during the genocide and who survived to it, continue to have a lower survival probability at least until their fifth year of life. This means that, even after excluding the direct consequences of the genocide (deaths occurred during the genocide), the impact on children's probability to survive remains strong. In other words, the genocide creates a fragile generation of children that needs to be safeguarded well after the end of the critical stage of the emergency. So, even if massive (and not indiscriminate) emergency intervention is needed to limit human life losses during the critical phase, middle term follow-up intervention supporting the fragile generation is crucial too.

These last observation creates a ground for new and interesting research questions. If it is true that violent conflicts create fragile generations, our analysis has the limit of focusing only on a very essential health outcome (survival). What about other possible not yet analysed aspects of this fragility? For policy purposes, it would be very interesting to track the specific psychological or physiological effects on health, or the consequences for the attitude towards violence or empowerment of the exposed generations.

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