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The association between birth spacing and infant mortality: Evidence from Nicaragua

生育間隔與嬰兒死亡率的關聯性: 尼加拉瓜的實證

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### Abstract

This study aims to provide evidence on the association between the length of birth spacing and infant mortality, and the mechanisms by which the interval produces deleterious effect in child survival for the Nicaraguan experience. Using pooled data from the Demographic and Health Surveys of 1998-2011, the association is evidenced by using survival analysis. The results show that there's generalized deleterious effect of the shortest than 18 months interval for Nicaraguan mothers, while finding indication of the existence of maternal depletion syndrome and sibling competition. A larger effect prevails for teenage mothers that space their children closely, possibly due to her unreadiness for childbearing and childbirth. While, the lack of wealth of the household and the inaccessibility to public health care of which rural areas suffer from, can play a large role in enhancing the deleterious effect of the short birth interval through the boost of any form of sibling competition. Concrete policy implications should be segmented by age groups and respond to different capabilities of family responses to deal with the potential effects of a short-spaced pregnancy and childbirth.

Keywords: Birth spacing, infant mortality, causal mechanisms, Nicaragua, survival analysis

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關鍵詞:生育間隔,嬰兒死亡率,因果機制,尼加拉瓜,生存分析

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### **1** Introduction

Around 5.3 million children died in 2018 worldwide before reaching five years of age. Even more alarming is the fact that 75% of those children didn't even live past their first year<sup>1</sup>. Infant mortality is a health tragedy that has greatly affected the world, much more so before the current reproductive health care technological developments widely diffused. Over the past decades, the world has constantly reduced the rates of death of this young and entirely dependent group, only in the past twenty-years the infant mortality death has been cut by half, falling from 56.3 to 28.9 deaths per 1,000 live births. Nonetheless, the decrease of infant mortality has not been equal, as some regions are still falling behind in the reduction targets. Nearly 80% of the 4 million of the infant deaths of 2018 occurred in Sub-Saharan Africa or South Asia (49% and 30%, respectively).

On the contrary, in the latter half of the past century Latin America has had the best performance in reducing child mortality, with most countries reducing at least 20% of their child mortality rates, while others –including Nicaragua– had cut off the mortality rates among children in half from 1980-2000 (Ahmad, Lopez & Inoue, 2000). Although, in Nicaragua the greater results of the reduction of the infant mortality rate were seen during the early 80s and from the mid-1990s throughout 2009, averaging a 5% yearly consistent decline. Nonetheless, in recent years the rate of infant mortality in the country has continued to decline but at a very slow rate reaching a decrease of 0.6% to 1% decline yearly (World Bank, 2020).

On the other hand, the good historical performance doesn't hold as strongly when dissecting the comparison within the Latin American region. Nicaragua has the second largest infant mortality rate in Central America –and the 11<sup>th</sup> among Latin American countries– with a rate of 15.7 deaths of children belong one year old per 1,000 live births, i.e. 1.5% of Nicaraguan children will not live past their first year and 1.8% will pass away before turning five years old.

The target of the Sustainable Development Goals (SDGs) is to reduce by 2030 the child mortality to 25 per 1,000 live births, thus, the target has been met in Nicaragua since 2003. But the issue remains relevant because most of these deaths are preventable; under a free-public health care system and considering all the modern methods to prevent and treat diseases during early childhood the rate is still too high, even when compared to the Latin American average of 14 deaths per 1,000 live births. Among the main causes of

<sup>&</sup>lt;sup>1</sup> Statistics on number of infant deaths and infant mortality rates taken from the World Bank Data (2020).

infant mortality in Nicaragua, the Ministry of Health points out the following reasons: respiratory distress syndrome, sepsis, asphyxia, birth defects, pneumonia and severe diarrhea (Ministerio de Salud, 2008).

Nevertheless, all of these conditions and diseases have their own set of causes and triggers. Particularly, research has identified the largest causes in developing countries to be associated with malnutrition, as the lack of nutrients make children more vulnerable to infectious diseases, as well as quantity and frequency, namely mother's birth parity and child spacing, since those dictate how resources are spread within the household (Blau, 1986). Indeed, the World Health Organization (WHO) has expressly suggested waiting at least 24 months after a live birth to attempt another pregnancy, that is, an interbirth interval of 33 months or roughly 3 years. The recommendation pursues the objective of reducing the risk of adverse maternal, perinatal and infant outcomes (World Health Organization, 2007).

Considering the above, the Nicaraguan government has recognized in official documents and large-scale health strategy planning that young mothers and short birth intervals have a deleterious effect in child health and development, increasing their risk of death (Ministerio de Salud, 2007). Within the same document, the country-level strategy for sexual and reproductive health highlights the main pathways towards improving mothers and children's health: (1) Through sexual education, that leads to increase awareness of family planning and also increases teenage first sexual-act age; (2) through family planning and contraceptives, by actually meeting the unsatisfied demand and increasing the utilization rate; and through the improvement and expansion of health care services, such as increasing institutional births and the coverage of antenatal care.

Despite these guidelines, the issue lies on the lack of objective actions proposed nor taken. Sexual education remains a weak spot on the policy actions, as schools do not approach sexuality topics as properly and in-depth as it's needed by the Nicaraguan youth. In addition, there are many teenagers and young people that escape the policies as they are out of the educational system. There's no evidence, despite being recognized as a potential influencing factor in infant, child and maternal mortality, that sexual education in Nicaragua addresses the importance of birth spacing as a tool to reduce the risk of exposing both mothers and children to health hazards.

On top of that, there's also little evidence that supports the WHO claims of the deleterious effects of short or long birth intervals for the Nicaraguan case, nor there's evidence on the possible mechanism by which such association can be made. There is a

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lot to be said about whether advocating to promote an optimal interval provides a viable infant and child mortality reduction route. The former lies on the fact that the promotion of a healthy-recommended birth interval is a not so well understood public health intervention, consequently also heavily underutilized (Norton, 2005). The importance of proper birth spacing is widely known, but for the Nicaraguan case, is based on evidence for other countries and regions and has not been used as a policy tool. Nicaragua, thus, has an unrealized potential of tackling infant and child mortality through polices directed to increase the spacing between births and improving mothers and children health through healthier reproductive practices.

### 1.1. Problem

On one hand, infant mortality remains high in Nicaragua, when compared to the Latin American statistics, and most of these deaths occur due to preventable diseases and health complications. On the other hand, a significantly large proportion of Nicaraguan mothers choose to have children very shortly spaced apart (less than 18 months). At the same time, it's widely promoted by international organizations and backed on international research on the past century, that short child spacing does impact negatively the risk of death of a child and has deleterious health outcomes. Despite these broadly recognized propositions, there's little evidence for the Nicaraguan experience regarding how impactful and damaging, if at all, can the short birth intervals be for child survival outcomes; and also, what possible mechanisms would those effects be attributed to.

### **1.2.** Purpose

This study aims to provide evidence on the association between the length of birth spacing and infant mortality, and the mechanisms by which the interval produces deleterious effect in child survival for the Nicaraguan experience based on data from the Demographic and Health Surveys of 1998-2011. Particularly, this study's purpose is to analyze how child spacing along with a set of other possible incidence factors can increase or decrease the risk of death that a child is exposed to; and which mechanisms will these results be associated with. Ultimately, the results provided by this research also aim to serve as baseline information for the policy makers and contribute to better shape public and private interventions towards addressing the health tragedy that is infant and child mortality. By providing evidence on this matter, policy makers can better shape their strategies towards reducing infant mortality and increasing the welfare of Nicaraguan families.

### **2** Literature Review

The study of human fertility has followed, from multidisciplinary standpoints, the main dynamics of population transitions in the world. Those transitions, whether attained as responses to economic transformations at regional, country and household levels, or to technological innovations regarding family planning mechanisms, among other possible causes, have fueled scholarly discussion for decades. The truth is that in the past six decades, family size has fallen by half –from 5 to 2.5 children per family– in a worldwide trend (Darroch, 2013); and family planning, boosted as public policy for developed and developing countries alike, is more widely used and has improved parental decision regarding number and timing of fertility (Powell, 1995).

These factors introduce a new variable in the household fertility decision-making, child spacing. The consistent decline of family size expands the possibilities for families, that deal with the same fertility time constraint but want fewer children. Therefore, time spacing between births becomes a key issue, especially when these decisions have important consequences in the life outcomes of the members of the household.

### 2.1. Child spacing

Decisions regarding child spacing are also determined by heterogeneous characteristics that surround human behavior. In their comprehensive cross-country comparative study, Rodriguez, et al. (1984) found that birth intervals were determined by early behavior and socioeconomic differences that affect the reproductive process, especially for transitional societies, i.e. developed countries, such as education, through its incidence breastfeeding and contraceptive behavior; and age at the start of the interval, through aging sterility, ultimately ceasing the opportunity window for reproduction.

In regards to age, for the interval between the first and second child, older women were found to be more likely to space their births closer than younger women did (Wineberg & McCarthy, 1989). These reflect the pressure due to the fertility time constraint and the uses of contraception among younger women experienced in the last decades since Morgan and Rindfuss (1999) found that the relationship between early childbearing, parents having more children in their lifetimes and the subsequent rapid pacing of the births, weakened deeply in comparison to previous studies (Bumpass, Rindfuss & Janosik, 1978; Trussell & Menken, 1978).

Among other determinants for the spacing between children are the preferences regarding parental presence, specifically those in terms of breastfeeding. The prolonged

breastfeeding does not only benefit child nurture and development but is also the cause of lactational amenorrhea in breastfeeding moms (Smith, 1985). Additionally, breastfeeding practices are also subject to cultural traditions and societal influence, and as Smith (1985) also finds, the median of some countries in Southeast Asia and Sub-Saharan Africa between 15 to 30 months of prolonged breastfeeding. Thus, whether as byproduct decision of increased education or indirectly through cultural legacy traditions, longer periods of full-breastfeeding increase the spacing between births.

Returning to Rodriguez, et al. (1984), households in developed countries that have tools to highly control their fertility, evaluate the timing and spacing of early births; but the later ones may include pregnancies product of contraceptive failures. For which, the authors suggest accounting for socioeconomic variables such as housing tenure, race and religion. Nonetheless, there are also unaccounted for occurrences in the household that might lead to changes in child spacing. For instance, Winikoff (1987) suggests that unusual long intervals between births can be caused by disruptions to the family ecosystem, e.g. divorce, death of one of the parents. Or on the other hand, biological factors mainly associated with the mothers, such as predisposition to fetal loss, death of the previous child, abortions, and the mother's health on itself, are all associated with the variations in the length of birth intervals (Winikoff, 1987).

Considering the aforementioned determinants, families decide upon optimal length intervals but this decision has consequences in life outcome for the offspring and the household on itself. It's also argued that households may foresee these unwanted outcomes and modify their selection of spacing length. On the macro level, longer interval reduces the potential of child production when considering the fertility time constraint, thus reducing the rate of population growth. Alternatively, it can also impact the rate of growth of a population through its incidence on the average spacing between generations (Newman, 1983).

At the household level, larger spacing between children serves to reduce the family size (Hanushek, 1992), which is, in turn, a key factor in increasing the family resources allocated, such as investments in human capital and parental time, namely resource-dilution hypothesis of Blake (1981). Still, on the micro-level, longer child spacing increases the cost of raising children, when assuming economies of scale, i.e. children of close ages have similar needs that can be met more efficiently when attending their needs collectively (Newman, 1981, as cited in Newman & McCulloch, 1984). On

the contrary, according to Powell (1995), longer birth intervals can favor the recovery from financial difficulties, consumption smoothing and proper spending planning.

Furthermore, the spacing decision has not only consequences on how the income is allocated, but for starters, how is it made. For instance, in dynamic models of fertility, delaying the timing of first birth and closed spacing decisions reduce the effects of childbearing in the labor market, i.e. reducing the opportunity cost of having children, represented in household forgone wages, human capital investments and depreciation (Troske, 2013).

When the focus is changed to spacing outcome effects in children – while building on the resource-dilution hypothesis–, longer spacing determines the number of children clustered at a particular age group which prominently determines the allocation of household resources (Powell, 1995). In this regard, the more intensive investment in children can be coined to higher quality, specifically reflected educational performance variables of children with longer spacing intervals.

Nonetheless, the more immediate results of birth spacing are those related to the health adverse perinatal and infant outcomes. Research suggests that when births are spaced closely or too far apart, there's an increase in the risk of adverse perinatal outcomes such as preterm birth, low weight at birth, smaller size for gestational age and underweight (Conde-Agudelo, Rosas-Bermúdez, & Kafury-Goeta, 2006; Conde-Agudelo, et al., 2012).

For surviving offspring, regardless of these adverse predispositions, they are more likely to suffer from short- and long-term health consequences of the short birth spacing. Notably, most effects are associated with nutrition and weight. For instance, Rutstein and Johnson (2004) compared 24-29 months intervals to 36-41 months ones, finding a decrease in underweight of 29% for the long-spaced births. A year later, Rutstein (2005) elaborated on another outcome variable, nutrition, and found that shorter birth spacing has a clear pattern of more undernutrition. In this sense, his empirical research found that the association of chronic malnutrition with birth intervals was statistically significant in 6 out of 14 surveys that collected anthropometric measurements data, while the relationship of spacing with general malnutrition was found significant in 5 surveys.

Incidences in the quality of nutrition are of particular interest when they become persistent and its cumulative effects transform into long-term impacts on a child's health, i.e. stunting, low height-for-age parameter. When the relationship between longer spacing and lower risk of malnutrition is confirmed, it has been found that intervals of more than 36 months reduced stunting from 10% to 50% (Dewey & Cohen, 2007). This is a particularly pressing issue for the developing world, where leading risk factors are primarily related to communicable, maternal, perinatal, and nutritional conditions; in stark contrast with developed countries, where the risk is mainly associated with non-communicable diseases (Lopez, et al., 2006).

All of the above puts into perspective the deleterious effects on children either at one point in time or in the long-run of those who survive. But it is more important to consider that the persistence and worsening of these health –and other household's–impacts as a result of the length of the spacing on children might deeply reduce the odds of survival for children past certain ages. The triggering of all possible positive consequences of the length of birth intervals and reduction of family size, described above relies on the assumption of living offspring. In the end, reduction of child mortality is a developmental, human and ethical priority for the world's agenda, especially in the developing world, where the incidence of this phenomenon is still far from fully mitigated.

### 2.2. Infant and child mortality

Infant mortality has been a consistent historical world health tragedy. Volk and Atkinson (2013) estimated evolutionary data for infant mortality, finding that historically up until the beginning of the last century, around 27% of children failed to survive past their first year of life, while 47.5% did not survive past puberty. From the 1950s through the 1960s, child mortality rates in Africa were still similar to those depicted in the evolutionary estimates, around one of three children died before they reached five years of age, while in Asia one in four children failed to survive their fifth year of life (United Nations, 2019).

Nonetheless, mortality rates among children below 5 years of age have constantly declined over the past 70 years, though a sharp contrast can be seen in the trends of reduction of child mortality for high- and low-income regions. Not only on this indicator but as a characteristic of the development of countries in the past decade, inequality and poverty fueled the global needs for a joint effort on improving welfare around the world. The latter resulting in early global initiatives such as the Universal Declaration of Human Rights (UDHR) or research-based policy-making of the development decade in the 1960s (Hulme, 2008).

The more substantive shift towards prioritizing world problems like child and infant mortality came in the 1990s, through the human development approach that permeated into international organizations. The earliest discussion of what provides the basis of this approach is Amartya Sen's conceptual framework on capability approach – contained in his lecture from 1979 through 1987<sup>2</sup>–, focusing on people as 'ends' of development, i.e. a developmental theory people-centric (Alkire, 2010). This prominent school of thought continued to grow and develop their approach, concepts and measures, but a premature impact is found in the publication of the 1990's United Nations Development Programme (UNDP) first Human Development Report (HDR). The report called for global actions to enlarge people's choices and forming human capabilities, rather than centering on traditional economic measures. HDRs also cross-over developmental topics with humanity's pressing issues such as gender, natural resources, sustainability, climate change, democracy and inequality.

Also, in 1990 the World Summit for Children took place, which was the largestto-date gathering of heads of state, to adopt policies to improve children's wellbeing. The target agreed on was to reduce the under-five child mortality rate by one third or to 70 per 1,000 live births, whichever yielded a lower indicator (United Nations Children's Fund, 1990). Meanwhile, international donors, such as USAID's Child Survival Initiative, set broad strategies for achieving certain targets on reduction of child mortality; mainly through better immunization coverage, oral rehydration therapy, boosting health and nutrition of mothers and children, and by reducing high-risk births (Ahmad, et al., 2000).

All of these steps, along with the increasing global partnership sentiment, paved the way to the world's biggest commitment among states and multilateral agencies to reduce poverty and improve multidimensional wellbeing, the Millennium Development Goals (MDGs) in 2000. The MDGs included infant, child and maternal mortality targets as main goals, despite them being absent from preceding material "We the Peoples - The Role of the United Nations in the 21st Century" and the concerns from the Vatican and conservative Islamic states (Hulme, 2008). The goal set was to reduce by two thirds the under-five child mortality rate in the period from 1990 to 2015, but by 2015 the rate was reduced by half, dropping from 90 to 43 deaths per 1,000 live births, failing to meet the MDG (United Nations, 2015a).

<sup>&</sup>lt;sup>2</sup> Alkire (2010) highlights Sen's 1979 lecture 'Equality of What?, 1985's 'Well-being, Agency and Freedom', and 1987's 'Commodities and Capabilities' and 'The Standard of Living'; which cover the starting point of the capability approach, more philosophical development to it, and linkages to economic development.

Also, by 2015, the global partnership goals evolved into the Sustainable Development Goals (SDGs), where child mortality became an indicator instead of a goal on itself. The now renewed target plans to reduce neonatal mortality to 12 per 1,000 live births and under-5 mortality to 25 per 1,000 live births by 2030 (United Nations, 2015b). In 2017, children 0 to 5 years old died in the same proportions –5.4 million– as older age groups did: 5 million of the 65-69 years old group, 5.3 million from the 70-74 years old group or the 85-89 years old group with 5.3 million deaths (Institute for Health Metrics and Evaluation, 2018). These facts along with the failure to reduce the original millennium goal, the new multiple goals reflected on the SDGs and the downgrading of child mortality from a pressing issue to yet another indicator, places more uncertainty on the effective achievement of this target in the foreseeable future.

### **2.3.** Child spacing effects on infant and child mortality

The spacing of births has a long-documented association with infant and child mortality outcomes, in fact, the earliest studies date of almost a century ago and already present the notions of the deleterious effect of shorter birth spacing and optimal interbirth intervals (Stevenson (1923) and Hughes (1923) as cited in Hobcraft, McDonald & Rutstein (1983)). The research on this effect has been approached from different disciplines, such as population studies, medicine, sociology and economics, to name a few. Nonetheless, it has moved to mainstream knowledge as a result of international organizations endorsing this proposal as a policy recommendation.

Particularly, the World Health Organization (WHO) has expressly suggested waiting at least 24 months after a live birth to attempt another pregnancy, that is, an interbirth interval of 33 months or roughly 3 years. The recommendation pursues the objective of reducing the risk of adverse maternal, perinatal and infant outcomes (World Health Organization, 2007). Experts that participated on that 2005's technical consultation agreed on the notion of an the deleterious effect of short intervals, and concluded the following: (1) birth-to-pregnancy intervals of six months or shorter had a higher associated risk of maternal mortality; and (2) birth-to-pregnancy intervals shorter than 18 months had a greater risk of infant, neonatal and perinatal mortality, lower weight at birth, being smaller for gestational age, and pre-term delivery (World Health Organization, 2007).

Multidisciplinary studies have attempted, for almost a century, to provide evidence on this long-running seemingly strong negative relationship. Although, the exploration of formal channels or mechanism by which child spacing can have deleterious effect on maternal and child health and survival started in the mid-1960s. In spite of the multiple causal mechanism assessed over the years, the literature usually coincides in three main mechanism for the "short intervals-infant mortality" relationship: a) maternal depletion syndrome, b) sibling competition and c) insufficient breastfeeding (Conde-Agudelo, et al., 2012). Most of the studies also include confounding factors, that if controlled, can isolate the effect of child spacing. Some of these factors include socioeconomic conditions and previous birth outcomes, to name a few.

### 2.3.1. Causal mechanism of effects of spacing in infant mortality

### a) Maternal depletion syndrome

This mechanism was first discussed by Jelliffe and Maddocks in 1964, although, their early proposal did not include child spacing. The authors introduced the notion that women who are in a continuous cycle of reproduction, synthesizing fetal and placental protein and producing breast milk non-stop, might affect the weight of their children and her health status (Jelliffe & Maddocks, 1964). When including child spacing, a mother with constant pregnancies and short birth intervals does not get the chance to recover and replenish her nutritional values, therefore increasing the odds of pregnancy losses and having low birth weight children (Hobcraft, et al., 1983).

Among some of the representations of maternal depletion that affect mother's and child health outcomes are the exhaustion of energy and protein resulting from short interpregnancy intervals (King, 2003) and the risk of folate insufficiency, which parallelly, impacts their children's hazard of neural tube defects, retarded intrauterine growth, and preterm birth (Smits & Esseds, 2001). DaVanzo, et al. (2008) found that the survival outcome of the pregnancy amplifies the maternal depletion syndrome, that is, live births or stillbirths are more depleting than miscarriages or abortions. Additionally, live births are usually followed by periods of breastfeeding which further depletes maternal physiological and nutritional stores.

Children with low weight at birth have a greater risk of mortality than those with an average normal weight and the group differences are accentuated for those with socioeconomic disadvantages (McCormick, 1985). Regardless of technological improvements and increased use of modern medical methods, the change in survival for very low birth weight –those who weighed less than 1500g at birth– had barely improved between 1990 and 2002 (Fanaroff, et al., 2007). The situation worsens for developing countries. Children severely growth-restricted and those who were born preterm are at higher risk of perinatal death, primarily of complications that in are not fatal in developed countries, but which care access is limited and difficult for developing countries (Kramer & Victora, 2001). Since women that already have inadequate food intake and are unable to adjust their energy expenditure to lower levels are the most likely to suffer from maternal depletion (Conde-Agudelo, et al., 2012), low-income mothers are more vulnerable to be affected by this mechanism.

### b) Sibling competition

Another potential mechanism whereby a short or long interval may decrease the odds of survival is for the children in the family is sibling competition. This occurs in two instances: on one hand, when two or more children, are closely spaced, they will grow up close in ages which may lead them to compete for family scarce economic resources and parental care (Conde-Agudelo, et al., 2012); on the other hand, when children are spaced longer, older siblings may take precedence in taking the limited available food supplies or resources of the family (Hobcraft, et al., 1983).

This mechanism is usually tested by including the survival status of the preceding child or the index child and the preceding or subsequent interval. Nevertheless, caution is advised when assessing the survival of the preceding sibling and its impact on the length of the interpregnancy interval. If the precedent child dies in infancy, the interval to the next birth could be shortened by an involuntary cessation of breastfeeding and temporal infertility associated with lactational amenorrhea and/or the mother's grief (Sweemer, 1984) and desire to replace the deceased child (Conde-Agudelo, et al., 2012). Thus, providing evidence on this effect has been a challenge, both theoretically and empirically, since arguably this effect is prompt to be harsher in poor families in developing countries in contrast with middle- and high- income households.

### c) Breastfeeding-pregnancy overlap

This effect is also depicted as a type of competition among the precedent child and the new shortly spaced pregnancy. The new pregnancy induces earlier weaning on the precedent child, with consequent deleterious effects on his survival (Hobcraft, et al., 1983). Additionally, when breastfeeding–pregnancy overlap, the intakes per feeding are lower and the weight gain associated with breastfeeding nurture decreases in the corresponding for one month (Marquis, et al., 2002). Another possible effect of the overlapping breastfeeding and pregnancy is change in the composition of breast milk, particularly affecting immunity nutrients on it, such as lysozyme concentration, lactoferrin concentration and Immunoglobulin A (Marquis, et al., 2003).

Interestingly, the breastfeeding-pregnancy overlap and the short interval can be related in two ways that lead to the detrimental effect on the survival of children. For instance, the short interval can lead to early weaning and lower quality breast milk, which affects the survival odds of the preceding child; and on the other hand, the maternal decision on the length of breastfeeding also affects the birth interval through lactational amenorrhea inducing effects on mortality of both the preceding and subsequent child.

#### d) Alternative mechanisms

The mechanisms described above are among the most commonly used to support the detrimental effects of child spacing and child mortality. Nonetheless, these are far from being the only possible channels of impact in the odds of survival. Many studies opt for controlling for socioeconomic variables or confounding factors, such as income, which is a determinant of nutritional intake for the family; parents' education, particularly mother's, as it represents maternal use of contraception, awareness of child care, and so forth (Sweemer, 1984; Hobcraft, et al., 1983; Boerma & Bicego, 1992; Forste, 1994; DaVanzo, et al., 2008).

There's a gap in the literature that should be addressed more, which is interpregnancy intervals and whether they change according to the outcome of the preceding pregnancy, i.e. whether it resulted on live birth, stillbirth, miscarriage, or induced abortion (DaVanzo, et al., 2008). These outcomes are key to properly identifying the dimension of the mechanism described above. For instance, non-live outcomes should be less depleting that a full-term live pregnancy that is followed by breastfeeding, also if the pregnancy outcome is not a live birth, there's no immediate sibling to compete or to overlap breastfeeding and pregnancy with.

Lastly, there are some more difficult to measure the mechanism of impact such as the psychological and emotional drain of mothers responsible for caring of a large family with scarce resources. These mental and emotional health effects or quality-of-life effects caused by larger families and shorter intervals are not measured nor included but maybe impacting maternal physical and psychological reserves (Winikoff, 1987).

### 2.3.2. Empirical evidence of the mechanisms in the literature

Previously it's been indicated research that has pioneered hypothesizing and/or proving the causal mechanism that leads short or very long intervals to result in child

mortality. In this subsection, research that provides evidence or questions the existence of such mechanisms is summarized.

In regards to the maternal depletion syndrome mechanism, the effects of intervals in child mortality are greater for the shortest intervals, which are the ones that give the littlest time for mother's recovery (DaVanzo, et al., 2008; Rutstein, 2005; Sweemer, 1984); while King (2003) confirms that depletion syndrome through the analysis of deficiencies in micronutrients, iron and folate in closely spaced pregnancies, which are at high risk of mortality, lastly, Boerma and Bicego (1992) provide evidence of both, maternal depletion and breastfeeding-pregnancy overlap causal mechanism, of how shortly spaced births affect mother's physiology and nutritional status, which in turn impacts the odds of child survival.

Regardless, other empirical studies have not found evidence for maternal depletion syndrome or inconsistent proof. For instance, Dewey (2007) found that longer birth intervals are associated with lower child malnutrition in some populations analyzed, but not all of them. She also found little evidence of the inverse relationship, shorter intervals with higher child nutrition, which remarks about the lack of statistical significance of this relationship. Winkvist, et al. (1994) propose that how the nutrients get assigned to mothers or children is affected primarily by the mother's nutritional status rather than the child spacing. In this case, the authors suggest than endorsing longer intervals is not effective enough, and that a better approach is to support nutrition for women at all stages of her reproductive cycle.

As for the sibling competition hypothesis, DaVanzo, et al. (2008) and Sweemer (1984) found supporting evidence of this effect. Particularly the former found for the postneonatal period and childhood, the detrimental effects of close spacing are greater if the preceding child is still alive at the time of the subsequent child's birth –giving them room to compete for family resources– than if the preceding had died.

On the contrary, Boerma and Bicego (1992) controlled for survival status of the preceding child and found that it does not reflect on increasing the effects of close spacing on mortality when the previous child is still alive, i.e. a supposed 'competition' environment. Therefore, they conclude that the sibling competition mechanism is not evidenced nor operative at all, but rather that the effects of familiar mortality risks are stronger than the competition mechanism. Thus, prenatal mechanisms are more relevant than postnatal ones, when explaining the causal nature of the child spacing effects on child mortality.

For the breastfeeding-pregnancy overlap, most studies argue for an effect of early cessation of breastfeeding, weaning, as a result of a closely spaced conception (Hobcraft, et al.,1983, Sweemer, 1984; Forste, 1994; DaVanzo, et al., 2008). Despite medical literature assessing the impact in breast milk nutrients and child weight, there's little direct evidence of these quality effects affecting child mortality through a shorter birth interval (Marquis, et al., 2002; Marquis, et al., 2003).

Research that aims to untangle the relationship between child spacing and child mortality is very broad, expands through disciplines and deals with complex multilateral relationships between variables and mechanisms. Experts fail to agree on the existence of certain effects and channels, as the discussion grows into different realities. In fact, human fertility is subject to many unmeasurable variables –culture, religiousness– and fundamental differences –developed and developing countries, health care systems– than unanimity upon the subject is not expected.

### **3** Data and Methods

### **3.1.** Data

To study the impact of the interbirth interval on infant mortality among Nicaraguan families and the mechanism by which it affects, data from the Demographic and Health Surveys (DHS) were used. The DHS started in 1984, building on the experience of its predecessors the World Fertility Surveys and the Contraceptive Prevalence Surveys. To date, it has become a widely spread, nationally representative, and comparable household data source, allowing to document demographic dynamics, such as fertility, family planning, maternal and child health in intervals of approximately five years (Fabic, Choi & Bird, 2012).

Nicaragua first enrolled in the DHS program in 1997, after years of battling the economic downfall inherited from the Sandinista government and a decade of civil conflicts, during its third phase primarily financed by the United States Agency for International Development (USAID). Thereafter, the country has continuously teamed up with different international organizations to gather the data until the last DHS developed in 2011-2012 (see Table 1). No more data has been available ever since, possibly due to ambiguous and hostile policies of the Ortega government towards foreign aid agencies – that led to the departure of the United Nations Development Program (UNDP) in 2016– and the halting of funds due to the 2018 sociopolitical crisis (Martí, 2019).

Data	Period	Primary donor	Source	Selection criteria	Sample size	
Data				Selection criteria	Households	Women
	1997-1998	USAID	DHS Program	<ul><li>Households:</li><li>National census-</li></ul>	11,528	13,634
Demographic	2001	USAID		<ul><li>based sampling</li><li>Women:</li><li>Resident of the</li></ul>	11,328	13,060
and Health Survey (DHS)	2006-2007	UNFPA <sup>1</sup>	World Bank	<ul> <li>household</li> <li>Fertile age: 15 to 49 years old</li> <li>Under five years</li> </ul>	17,209	14,221
	2011-2012	The Global Fund	INIDE <sup>2</sup>	old offspring of each selected women	21,960	15,266

 Table 1: Nicaraguan DHS (1998-2011) general description

<sup>1</sup> United Nations Fund for Population Activities

<sup>2</sup> National Institute of Information for Development (Nicaragua, Spanish acronym)

Data on each DHS is cross-sectional; thus, the datasets were pooled to have a more comprehensive sample. The main dependent variable, infant mortality, is defined as a binary variable that depicts the occurrence of under-one-year old death. The DHS only gathers important nutritional and live outcomes variables from alive children under five at the time of the interview but reports the history of age at death for each woman's offspring. Therefore, in this study, all chosen variables that report on child-specific aspects will be those that cover the full record of offspring and not those later expanded in the dataset. On the contrary, the main independent variable will be defined as the interbirth interval (IBI), i.e. the time measured in months from the childbirth of the preceding child to the birth of the index child. This is mainly because there is no information on each pregnancy's duration, thus, other time measures such as birth-to-pregnancy (recommended by the WHO (2007)) cannot be obtained; nor there's data on the outcome of each pregnancy besides live birth, thus, neither the inter-outcome intervals can be found (used by DaVanzo, et al. (2008)).

To be included in the empirical model, the IBI was coded into 4 categories: firstborn, index children born first (inapplicable for the calculation of IBI); and IBI groups of: less than 18 months, between 18 to 35 months, and more than 36 months. These categories reflect on previous literature findings regarding the deleterious effects of short (less than 18 months) intervals (WHO, 2007).

Additionally, the selection of covariates serves two purposes: (1) control for confounding factors that may also explain infant mortality; and (2) address the objectives of assessing the existence and direction of effects of the causal mechanisms described in *Section 2.3.1.*, such as: maternal depletion and sibling competition for Nicaraguan

families. Nonetheless, one of the causal mechanisms that it's not possible to discuss due to the scope of the DHS data is the breastfeeding-pregnancy overlap. As mentioned above, DHS data only follows children that are reported alive, living at the household and younger than 5 years old at the time of the interview. Thus, information on breastfeeding practices, nutrition, immunization and pregnancy durations are not available for the main interest group.

Covariates are split into: a) index child-specific and referenced variable: interbirth intervals, gender of index child, singleton birth (single or multiple), birth order, and death of the preceding child; b) mother specific variables, such as: the highest level of education reached, mother's age, whether or not the mother has experienced a miscarriage; and c) household variables: a measure of the wealth of the household, number of adults living in the household and distance to health services. Additionally, by-groups specifications on: mortality of previous child, mother's age, birth order, household wealth and area of residence will be used to explore and discuss the empirical evidence of causal mechanisms and assess which effect prevails over the other possible channels.

These variables can be grouped by the mechanism each address, in this sense, variables such as IBI<18 months and the death of preceding child could reflect on the mother's depletion; and the sibling competition can be evidenced in variables such as birth order and multiple births. Lastly, to account for the effect of previous miscarriages or interruptions, binary variables for mothers that have had each of these occurrences were created. Although it is not possible to link these events to each child record, it still provides valuable information on the maternal reproductive and health history.

For the most part, the selection of these variables responds to a synthesis of the previous empirical literature and the viability of finding them in the DHS datasets. Primarily, the models followed are DaVanzo, et al. (2008), Fotso, et al. (2013) and Becher, et al. (2004). All variables are described in detail in Table 2 below.

Variable	Reference	Levels	Description
Index child-level variabl	es		
Interbirth interval (IBI)	DaVanzo, et	First born, <18 months, 18-35, 36+	Difference in months from the day of birth of the preceding child to the birth date of the index child
Infant mortality of index child	al. (2008)	Binary; 0=for surviving children, 1= died	Whether index child died while being under-one year old or survived (infant mortality criteria)
			(Continued)

Variable	Reference	Levels	Description		
Index child-level variab	oles				
Infant mortality of preceding childDaVanzo, et al. (2008)		Binary; 0= if preceding child survived, 1= died	Whether preceding child experienced infant mortality or survived		
Sex of the index child	Fotso, et al.	Binary; 0= male, 1= female	Sex of the index child		
Singleton birth	(2014)	Binary; 0= singleton birth, 1= multiple birth	Whether the birth was singleton or multiple		
Birth order	Becher, et al. (2004)	First born, $2^{nd}$ to $4^{th}$ , $\ge 5^{th}$	Categories that group children by the order in which they were born		
Mother-level variables					
Mother's education	DU	No schooling or primary; and secondary, tertiary or higher	Highest level of education reached by the mother		
Mother's age at birth	DaVanzo, et al. (2008)	<18, 18-35, ≥36	Mother's age at the time of birth of the index child measured in years		
Miscarriages	Proxy to the effects discussed in DaVanzo, et al. (2008)	Binary; 0= didn't have one, 1= has had a miscarriage/interruption	Whether the mother has had a pregnancy interruption (DHS 1998 2001) or a miscarriage (DHS 2006- 2011)		
Household-level variab	les Z				
Wealth index	Proxy to the one used by Fotso, et al. (2014)	Low= DHS WI<0, Middle= DHS WI of 0 to 2, High= DHS WI>2	Categories created out of a composite measure of household's cumulative living conditions following the DHS Wealth Index methodology.		
Adults living in the household		Alone, adults=1; accompanied by 1, adults=2; accompanied by 2+, adults>2	Number of adults (age>18) living with the mother in the household.		
Remoteness to health services	Proxy to the one used by Becher, et al. (2004)	Binary; $0=$ not far, $1=$ for reported to be far	Approximation using reported "remoteness" of health services in questions regarding usage of health care.		
Variables for by-groups					
Area of residence	Fotso, et al. (2014)	Binary; 0= urban, 1= rural	Whether the household is located in an urban or rural area		

### Table 2: Description of variables used in the empirical model

### **3.2. Empirical methods**

### 3.2.1. Survival analysis

An analysis of the promptness of a child to die before reaching one year of age due to the influence of the IBI and a vector of covariates could potentially be measured by using a logistical regression or a probabilistic approach. The issue is, then, we would only be measuring the likelihood of an event to occur subject to its control variables. A variable such as infant mortality does not only consider that the event –death– happens, but also when it happens. Thus, timing matters because there's a time frame of 12 months for it to be considered that a subject suffered from an 'infant death' and not any other category. Another crucial factor being, the fact that the mortality of children accelerates dramatically the closer it is to their birth date. This is evidenced in the fact that large percentages of children that died under the category of infant mortality are concentrated in neonatal mortality (within 28 days postpartum) and much more so in the very vulnerable group of perinatal mortality (within 1 week after childbirth). Thus, the function for the survival of these children is a non-linear function highly concentrated within the very first periods, behavior that is best captured by a survival analysis approach.

When the outcome variable of interest is the time until a specific event takes place, the statistical analysis can be done using the non-parametric and parametric estimations contained in the so-called survival analysis. Therefore, the dependent variable now has two important components: time, i.e. time elapsed from the beginning of the study until the event occurs or the study ends; and the other part is the event, that is, the indication that the individual studied had the experience of interest (Kleinbaum & Klein, 2010). These components are usually defined in survival analysis as the "survival time", for the time to event, and "failure" to the occurrence of the event; terms derived from the initial heavy influence of biostatistics in these methods that usually contemplated a survival time to death, diseases or other negative life outcomes (Kleinbaum & Klein, 2010).

Regarding one of the components of analysis, the event, there are different classifications for it. For instance, there's single events, i.e. those that account for duration for one event for each studied unit. Usually these events are assumed to be absorbing, i.e. can only happen once. In contrast, there's also the case of multiple events, which can be: (1) of multiple types, that is, different and absorbing events; and (2) recurrent events, when the same event is studied in repeated occasions (Skrondal & Rabe-Hesketh, 2004).

Additionally, following Hosmer, Lemeshow & May, 2008, while analyzing the other important component, 'survival time', it's key to understand the issue of "censoring". As a time measurement, the survival time has to properly define and count units of time elapsed from a beginning to an ending point. In the process, the observation of time might become incomplete, issues which are called censoring and truncation. The authors describe that an observation can be 'right-' or 'left-censored', the former, occurring when the 'time' finished before the 'event' or outcome of interested has occurred; and the latter, happens when, on the contrary, the event of interest has already happened when the observation begins. In regards to truncation, observations are incomplete because of the design of the study selection process. It's possible to encounter left truncation, also known as delayed entry, when the time to observe an individual is deliberately delayed; as well as, right truncation or length biased sampling, when all the studied population has experienced the event of interest and was selected for the analysis precisely because of that way before the study starts. The estimations contained in survival analysis can be divided into two main methodologies:

### a) Non-parametric estimations

Due to the particular issues described above, i.e. censoring and truncation, standard descriptive statistics will not properly estimate the parameters. Thus, Hosmer, Lemeshow & May (2008) suggest finding the cumulative distribution that can generate statistics in line with the interest parameters. This measurement is found in the survival function, denoted as S(t) = Pr(T > t), that expresses the probability that an observation's survival time (T) exceeds a specific point in time t. This is the most common measure, as the majority of studies are interested in subjects not to fail (e.g. live), rather than experience the failure (e.g. death), although focus on failure it's also possible by using the hazard function.

Generally, the Kaplan-Meier survival curves are used to estimate the survival probability, since it considers all the available information from the observations, censored or uncensored. Its functional form is given by Equation 1:

$$\widehat{S}(t_{(f-1)}) = \prod_{i=1}^{f-1} \widehat{Pr} \left( T > t_{(i)} \, \middle| \, T \ge t_{(i)} \right) \tag{1}$$

This definition indicates that the estimator derivates from the products of the sequence of conditional survival probability past the failure time  $(t_{(f)})$ , thus aiding to observe the shape of the survival function. The Kaplan-Meier estimator allows each

observation to contribute information while they are under the status of "surviving", those who experienced the event or are right-censored provide information for the at-risk group and later the former sums to the number of observations that failed.

Once the survival probability has been observed, it's key to determine whether or not the Kaplan-Meier survival curves are proportional or statically equivalent for relevant groups originated from the set of covariates, particularly, those that depict effects that are believed to be related to the survival of the study units. Specifically, it's important to measure differences among groups in order to assess the validity of including those variables in the final model. In this sense, common statistical test such as two sample hypothesis testing or the rank-sum test, will not yield proper estimations when dealing with censored data observations.

Hence, it's suggested to use the long-rank test, which builds on the survival curves (Kaplan-Meier) to provide evidence on differences at the population-level. Thus, the log-rank test runs a sample-wide  $\chi^2$  test to compare the curves, by comparing per categories the cell counts of observed and expected events over all failure times. Ultimately, the long-rank test helps to test the null hypothesis that there is no difference between two or more survival curves (Kleinbaum & Klein, 2010).

In addition to the KM survival curves, there are alternative estimators, the main one being the Nelson-Aalen one that instead of using the estimator S(t) defined in Equation 1, it developed one based on H(t). This estimator is also known as the cumulative hazard function in survival analysis and its graphical representation would emulate that of the survival function but in opposite direction, since, as H(t) or the cumulative hazard increases there will be a decrease of the same magnitude in the survival function S(t). The estimator is given by Equation 2:

$$\widetilde{H}(t) = \sum_{t_{(i)} \le 1} \frac{d_i}{n_i}$$
(2)

Where H(t) is the cumulative hazard, given the observed deaths  $d_i$  and those at risk of dying  $n_i$  at time t. Therefore, for large size of risk-to-events ratios, the survival functions found by both of the estimators will not yield great practical differences.

#### b) Semi-parametric estimation: The Cox proportional hazard model

The non-parametric methods described above cover a variety of methods within the so-called univariate analysis. Nonetheless, survival analysis can also exploit the timeto-event analysis with a set of variables that are thought to affect the occurrence of the event. Indeed, strong theoretically or empirically associated variables should not be omitted and should be treated as potential confounders. Thus, more complex analysis is only feasible through a multivariate analysis, namely a parametric regression model under the time-to-event framework.

One empirical method that serves this purpose is the Cox proportional hazard model, proposed by Cox (1972) as an expansion on the work of Kaplan Meier while incorporating regression parameters, that is, including explanatory variables that provide coefficients on the basis of a time function. Following Kleinbaum & Klein (2010), the Cox proportional hazard model can be defined as:

$$h(t,X) = h_0(t) e^{\sum_{i=1}^{p} \beta_i x_i}$$
(3)

Where the first term at the right of Equation 3,  $h_0(t)$ , represents the baseline hazard which considers time (t), but not any effects on the X vector (explanatory variables). Meanwhile, the second term  $e^{\sum_{i=1}^{p}\beta_i x_i}$  is the exponential expression, it addresses the covariates but not the time, since X's are assumed to be time-independent. This assumption is the proportional hazards which ultimately proposes that a change in an individual's explanatory factors induces a proportional change in his/her hazard rate. (thus, the multiplicative relation).

Among the strengths of the Cox proportional hazard model are: (1) it offers a robust estimation, i.e. the results will be consistent with those of the correct parametric model, which in turn, it's hard to establish the appropriate model; and (2) the Cox PH model relies on the introduction of the hazard ratio (HR) to measure the risk of exposure to the event, as described by Hosmer, Lemeshow & May (2008). Therefore, a hazard ratio that's equal to 1, will indicate a non-existing effect; a hazard ratio under 1 expresses a reduction in hazard; and finally, a HR greater than the unit means an increase in the hazard of exposure to the event. Additionally, the Cox PH model also allows to compare outcomes of hazard among groups, functioning as a "relative-risk" ratio when interpreting binary variables, for example.

Finally, to validate the use of the Cox PH model the assumptions of proportional hazards have to be assessed. In other words, assessing during postestimation the goodness of fit through hypothesis testing for relevant predictors. Majorly used, the Schoenfeld residuals, help provide a statistical test for covariates, both individually and globally, and whether they are or not related with the survival time. Testing for a null hypothesis of

proportional hazard, the  $\chi^2$  statistic will indicate the rejection or non-rejection of the null hypothesis described above.

#### c) Extended Cox proportional hazard model for time-dependent variables

When the test for the independence of predictors with the survival time fails to reject its null hypothesis, those predictors are concluded to be time-dependent. This invalidates the proportional hazard assumption for the Cox PH model. When encountering this situation, the Cox model can be extended to include terms of interaction between the time-dependent variable and a specific function of time.

In a similar way to the Cox PH model definition contained in equation 3, the extended model also includes a baseline hazard denoted by  $h_0(t)$  multiplied by an exponential term. Nevertheless, according to Kleinbaum & Klein (2010) the exponential function for the extended model now introduces the recurrent time-independent covariates  $X_i$  and the time-dependent covariates  $X_{j(t)}$ , seen in the right-hand side of Equation 4, while all predictors at time *t* are denoted by X(t) on the left-hand side of the equation:

$$h(t, X(t)) = h_0(t) e^{\left[\sum_{i=1}^{p_1} \beta_i x_i + \sum_{j=1}^{p_2} \delta_j x_j(t)\right]}$$
(4)

The authors also point out the vital assumption of the extended Cox model being that the effect of a time-dependent variable  $X_{j(t)}$  on the survival probability at time *t* depends on the value of the predictor at that same time *t*. Thus, the model provides only one coefficient for each time-dependent variable, depicted in  $\delta_j$  of equation 4. The statistical significance of the interaction term between a time-dependent covariate and the function of time represents the violation of the proportional hazard assumption for that specific covariate.

### d) Parametric estimation: The Weibull model

The main characteristic of parametric survival models is that they assume a known distribution for the survival time. Parametric models are preferred when an assumption on the distribution is feasible and estimated parameters can fully specify the survival and hazard functions.

Among them, the Weibull model is the most popular one as it is deemed to be more flexible, while the hazard function remains simple as it only rescales t to a fixed power (Kleinbaum & Klein, 2010). In addition, the Weibull model is the only parametric model that is able to hold both accelerated failure time (AFT) assumption and the proportional hazard (PH) assumption. Thus, making it suitable for modelling data with hazard rates that either increase or decrease over time. The hazard function under the Weibull model is given by the form:

$$h(t) = \lambda p(t)^{p-1} \tag{5}$$

Where  $\lambda$  is reparametrized in terms of predictor variables and the newly added parameter *p*, also called the shape parameter, serves as the indicator of the shape of the hazard function. For instance, when *p*>1 the hazard function increases over time; when *p*=1 the hazard function is constant and it reverts back to an exponential approach, and lastly, when *p*<1 then the hazard function decreases as time goes on. It is from this added parameter that the Weibull model is considered of great flexibility, as it adapts to the behavior of the hazard over time rather than assuming it from the beginning.

#### **3.2.2. Empirical model**

Using the pooled data from the 1998-2011 Nicaraguan DHS, the two main components of the dependent variable, event and survival time, are defined as: event, binary variable for infant mortality (under-one year old deaths); and the survival time, as the age at death for under one year-old children and for surviving children, their age up until the cutoff of the event.<sup>3</sup> The event, infant mortality, is –according to the definitions observed in *Section 3.2.1.–* a single and absorbing type of event.

In regards to censoring, the model for survival analysis of Nicaraguan children subject to the interbirth intervals incurs in right-censoring. This happens for all children that indeed experienced the event, i.e. they passed away but where not accounted for since they were older than 1 year old. On the other hand, the nature of the data collected by the DHS also allows to discuss the existence of unobserved left-censoring. That is because mothers could effectively have had a non-live outcome in her reproductive history, such as terminations of pregnancy and miscarriages, although those are not recorded by the DHS data. Therefore, there are children that experienced the event before being born, thus not becoming a live birth, but this is also the reason why they are not observations on the DHS, losing this information completely.

Starting with the non-parametric estimations, the Kaplan Meier survival curves will be estimated by groups of IBI categories and placed in the same graph, this definition will aid in recognizing graphically the survival differences among categories for the IBI ranging from very short, short and recommended birth intervals. It's expected to find a

<sup>&</sup>lt;sup>3</sup> The rationale behind this is simulating a study that individually followed each child for a period of 1 year from their birth date. Thus, the variable "survival time" will stop counting after their first birthday for those who survived, as it simulates the study 'ending'.

lower survival function and curves for very short interval, while a higher survival probability for those with a recommended length of IBI. Moving towards a proper definition of the semi-parametric model, log-rank tests will be used in all proposed explanatory variables to assess the differences among groups and provide evidence to support the inclusion of those variables in the final model. Finally, on the non-parametric methods, the consistency of the behavior found in the survival curves will be assessed through the calculation of the hazard functions using the Nelson-Aalen estimator and its graphical representations.

In respect to the semi-parametric proposed empirical strategy, the Cox proportional model will be used as depicted in Equation 3. Adjusted to the covariates selected for this study, the first model will have the form:

$$h(stime, X) = h_0(stime) e^{\sum_{i=1}^{12} \beta_i x_i}$$
(6)

Where *stime* represents the survival time, X is a vector of explanatory variables only at the child-level that include: *IBI* (interbirth interval); *mortpc* (mortality of preceding child); *gender* (gender of index child); *birthord* (birth order); and *stbirth* (singleton/multiple birth). A second model, will add to the vector of explanatory variables the controls at the mother and households levels: *mothereduc* (mother's educational level); *miscarr* (has had a miscarriage); *WI* (wealth index); *totadults* (total number of adults living in the household) and *healthser* (remoteness of health services). Additionally, the following variables will be used to *motherage* (age of mother at birth); *area* (area of residence). After the estimation of the three models, the Schoenfeld residuals will be used to globally test each estimation, and thus, determine the proportionality of hazards.

If some predictors were found to have non-proportional hazard functions, i.e. to be time-dependent, the initial Cox model will be modified to the extended Cox PH model of the form:

$$h(stime, X(stime)) = h_0(stime) e^{\left[\sum_{i=1}^{12} \beta_i x_i + \sum_{j=1}^{12} \delta_j x_j(stime)\right]}$$
(7)

Where *stime* is the survival time, X(i) is the same vector of explanatory variables depicted in Equation 6 for all three models, and X(j) comprises variables found to fail to reject the null hypothesis of time dependency in that specific predictor, as an interaction term with the time function. Finally, the specification of the parametric model using the Weibull distribution will be given by Equation 5, where  $\lambda$  will be reparametrized in terms of the vector of covariates at child level for model 1, at mother and household level for model 2, and *t* will be, similar to the models above, the survival time. Additionally, the Weibull model will be used for by-group definitions of the variables described in Table 2, with the purpose of finding evidence on which causal mechanism is more impactful.

### **3.2.3.** Expected results

In line with the objectives of this study presented in Section 1.2., the methods described in this section aim to provide a basis to corroborate or deny the followings premises:

- There is a deleterious effect of spacing childbirth in less than 18 months for the Nicaraguan case, evidenced to a higher hazard rate for children born in this interval.
- Spacing childbirth in 18-35 months or more than 36 months (WHO recommended interval) is beneficial for children survival, it reduces the hazard rate for children born in these intervals.
- There is evidence of two causal mechanism found in the literature: maternal depletion syndrome and sibling competition: variables such as death of the preceding child, birth order and singleton/multiple birth find a higher hazard of infant death.
- The model can provide information on the most impactful of those mechanisms: there's a clearer effect of larger magnitude for one of the mechanisms.

### **4** Results

### 4.1. Non-parametric findings

Firstly, the distribution of variables depicted in Table 2 was found comparatively for the group that died while being under-one-year old and the group that survived past their first year of life. These differences provide initial insight on potential effects of different covariates across survivor and non-survivor groups (see Table 3 below).

Among the main effects that a priori we can observe while comparing groups, regarding our main independent variable, interbirth interval, for children who died as an infant the short intervals had a larger representative share of 28.45%, while in the survivor group only a 13.4% had had the shorter interval. Conversely, those that were born with a longer interval (more than 18 months) were more prevalent among the survivor group (56.1%) that among children that passed away before reaching one year of age (39%). On the other hand, with slight differences, those who died were primarily male, while the surviving children are primarily female. The death of the preceding child seems to be a

relevant factor as there is a 15% of children who died as an infant that also had their

previous sibling passed away, as opposed to a 5% in the surviving group.

		famili	ies, 1998-20	)11			
	All births		Infant mortality		Survived past 1 year		Log-rank
Variable			(Under 1 years old)		old		test
	N	%	Ν	%	Ν	%	p-value
Index child-level variables							
Interbirth interval (mont	,						0.000
First born	41,791	30.65	2,331	32.56	39,460	30.54	
<18	19,342	14.19	2,037	28.45	17,305	13.40	
18-35	41,687	30.57	1,867	26.08	39,820	30.82	
36+	33,527	24.59	924	12.91	32,603	25.24	
Sex of the index child							0.000
Male	70,743	51.69	4,126	56.56	66,617	51.41	
Female	66,127	48.31	3,169	43.44	62,958	48.59	
Singleton/multiple birth	,				,		0.000
Singleton	134,743	98.45	6,811	93.37	127,932	98.73	
Multiple	2,127	1.55	484	6.63	1,643	1.27	
Death of preceding child		L.	メ /口				0.000
No	129,223	94.41	6,163	84.48	123,060	94.97	
Yes	7,647	5.59	1,132	15.52	6,515	5.03	
Birth order	7,047	5.57	1,152	15.52	0,515	5.05	0.000
First born	41,360	30.22	2,204	30.21	39,156	30.22	0.000
$2^{nd}$ to $4^{th}$	67,671	49.44	3,399	46.59	64,272	49.60	
$5^{\text{th}}$ +	27,839					20.18	
	27,839	20.34	1,692	23.19	26,147	20.18	
Mother-level variables					]]		
Mother's education	-				/ =		0.000
No schooling/							
Primary	112,572	71.85	6,258	85.78	106,314	71.17	
Secondary/Tertiary	44,098	28.15	1,037	14.22	43,061	28.83	
Mother's age at birth (ye		20.15	1,057	1	5,001	20.05	0.000
<18	21,903	16.07	1,775	24.93	20,128	15.58	
18-35	108,000	79.22	5,034	70.70	102,966	79.69	
36+	6,425	4.71	311	4.37	6,114	4.73	
Miscarriages***		$\sim 11$	enach				0.000
None	125,630	80.19	5,336	73.15	120,294	80.53	
Has had at least one	31,040	19.81	1,959	26.85	29,081	19.47	
Household-level variables	,		,		,		
Wealth index							0.000
Low	87,391	60.2	4,555	67.94	82,836	59.83	
Middle	25,801	17.77	1,163	17.35	24,638	17.79	
High	31,970	22.02	986	14.71	30,984	22.38	
Number of adults	,						0.000
Only the mother	13,775	8.73	544	7.46	13,231	8.79	
Accompanied by 1	76,311	48.36	3,423	46.94	72,888	48.43	
Accompanied by 2+	67,712	42.91	3,326	45.61	64,386	42.78	
Remoteness to health serv		/ 1	-,		- ,		0.000
Not far	136,096	86.87	6,047	82.89	130,049	87.06	0.000
Far	20,574	13.13	1,248	17.11	19,326	12.94	
Variables for by-groups sp		10.10	1,2 10	1/11	17,520	12.77	
Area of residence	cericanons						0.000
Urban	62,021	45.31	3,039	41.66	58,982	45.52	0.000
Rural	74,849	43.31 54.69	4,256	58.34	70,593	4 <i>3.32</i> 54.48	
*** This variable cont			,				

 Table 3: Distribution of selected socioeconomic variables among Nicaraguan families, 1998-2011

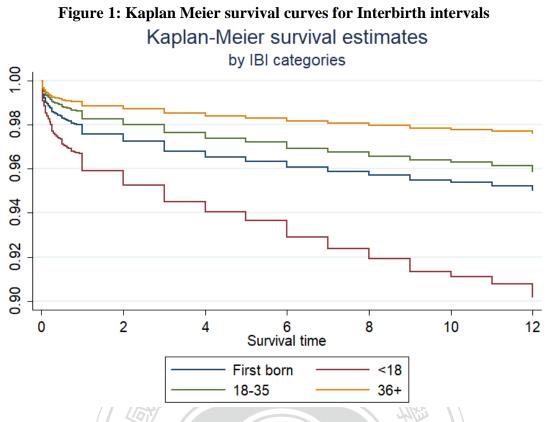
\*\*\* This variable contains information on pregnancy terminations and miscarriages, as the data contains years where abortion was legal and years after the illegalization of this practice.

Regarding mother-level covariates, these initial distributional data also point an important issue historical and current for Nicaraguan woman: poor educational attainments. Women who either attain a primary level of education or none at all account for almost a third of the women in the sample. Mother's education has been found to have a strong relationship with child mortality, as lower educated mothers have less access to sexual and reproductive health information, and this is observable in the Nicaraguan data, as there is a higher incidence of low educated mothers among those who died (85.78%). In respect to mother's age, a quarter of children that suffered from infant mortality where born to teenage moms (younger than 18 years old), as opposed to a 15.58% of survivors past one-year-old born to the same age-segment mothers.

Additionally, variables at the household-level show that the lack of readily resources negatively reflect the most among children that died. For instance, while examining the measurement of household wealth, it's noticeable the greater share of low-income families among infants that died (68%) than those who didn't. Conversely, high-income households can provide with better health care and resources thus being more prominently among the survivor group (22%) than poor families that have no way but to access primary care in the public system and have less resources to take better care of both the mother and the child before and after childbirth (15%). In second place, the results for the long-rank of each covariate are presented in the last column to the right of Table 3. The test leads to the rejection of the null hypothesis that there is no difference between two or more survival curves in all but one of the covariates.

Lastly on the non-parametric results for the survival of infants in Nicaragua, the graphical depictions of the survival and cumulative hazards by categories of interbirth intervals are presented. Figure 1 presents the Kaplan-Meier survival curves for infants according to the interbirth interval they have with their preceding sibling. In this sense, the KM curves clearly show that the lowest survival probability, and clearly separated from other categories, belongs to the children born in the shortest birth interval (less than 18 months); which also accelerates the decline of survival probabilities within the first periods of time to survival analysis.

Additionally, children born first have the second lowest probability of survival than those born of intervals of 18 to 35 months, or more than 36 months. This group being the one with the greater survival curve, which corresponds to the recommended longer intervals suggested by the WHO; all have a flatter behavior, i.e. do not present a constant reduction of survival probability as the children born to the shortest interval do.



Conversely, Figure 2 in Appendix presents the Nelson Aalen cumulative hazard curves for infants according to their preceding interbirth interval, which is the opposite function to the survival probability depicted in the Kaplan-Meier curves of Figure 1.

The cumulative hazard curves serve as a graphical check on the fit of a potential parametric model. Particularly, the "slope" of the hazard curve is steeper and accelerates more quickly for those children aged one month old or less. As mentioned above, the curves behaviors should be the complete opposite of the KM curves. Therefore, the group with the highest cumulative hazard is, again, children born to less than 18 months of interbirth interval; while first born, interbirth intervals of 18 to 35 months and notably, longer intervals (36+ months) have lower cumulative hazard rate.

### 4.2. Semi-parametric model findings

#### 4.2.1. Cox proportional hazards model

Before presenting the results for the Cox proportional hazard model, the consistency of the results of the survival analysis where tested through a linear probability model (See Table 9 in Appendix). The linear probability model shows consistent results the Cox PH model and the parametric model to be described in the next subsections. Where an additional month of interbirth interval decreases the probability of dying as an infant, while the other covariates also keep the direction of their expected effects.

The results for the Cox proportional hazard model are presented in Table 4. The first model, that only includes child-level covariates, finds the short IBI (less than 18 months) to have a 34% higher risk of dying as an infant that those who were born first, effect statistically significant at the 95% confidence level. Conversely, those born a 18-35 months or in the recommended intervals reduce the risk of dying in 39% and 63% compared to the first-born, respectively, at the 99% confidence level.

The deleterious effect of short interbirth intervals found in Table 4 might reflect on the mother's depletion syndrome, where mother's do not properly replenish her nutritionals stores for short spaced pregnancies, affecting the child's health outcomes thus increasing the risk of her children to die as an infant. Nonetheless, further testing and empirical exploration on the existence of this mechanism will be further discussed in the *subsection 4.3.2*.

Meanwhile, girls have a 19% lower risk of death compared to boys, and high order-born children, those born 5<sup>th</sup> or later in their family, have a 34% higher risk of infant death; and the hazard rate results for births that were multiple compared to those that were singleton show that children born to multiple birth have 334% higher risk of dying within one year of age as opposed to these that were a single-child birth. The former two effects described above might be indicators of the sibling competition mechanism, since older siblings may dominate most of the resources leaving younger-order siblings with limited access to those household resources; while having a same-aged sibling born at the exact same time means having a direct rival for the same kind of resources from the time of birth, e.g. mother's care and attention, breastfeeding time and quality.

Lastly for model 1, the death of the preceding child might imply a shorter preceding interbirth interval due to a 'replacement' effect (i.e. replacing the lost child with another very quickly) which goes back to mothers that might potentially suffer from maternal depletion syndrome. The results indicate that children that had their preceding sibling die have a 166% higher risk of dying as infant compared to those whose preceding sibling didn't die.

According to the proportional hazard test done through the Schoenfeld residuals, from these set of covariates sex of index child and multiple birth were found to be timedependent. Thus, the interactions between the natural logarithm of time and those covariates will be considered through the extended Cox proportional hazards model, showed in the following subsection in Table 5. Model 2 offers an insight on the effect of child-specific, mother-specific variables and household variables on the survival of Nicaraguan infants. This model maintains magnitude, direction and statistical significance of the effect of most covariates. For the case of the interbirth interval, children born from the shorter intervals have a 39% higher risk of having an infant death when compared to first born. Similar to model 1, the interval of 18-35 months and 36+ months, both indicate a lower risk of dying of 35% and 57% respectively in relation to children that were first born.

		Micaragu	а			
	(1)			(2)		
	HR	95% CI	P(PH)	HR	95% CI	P(PH)
IBI (Ref: first born)	/					
<18	1.34**	1.07-1.68	0.93	1.39**	1.10-1.76	0.53
18-35	0.61***	0.49-0.77	<b>L</b> 0.54	0.65***	0.51-0.83	0.86
36+	0.37***	0.29-0.46	0.11	0.43***	0.34-0.55	0.53
Sex of index child (Ref:	1			$\times$ $\setminus$		
male)	Y					
Female	0.81***	0.77-0.85	0.00	0.81***	0.77-0.85	0.00
Singleton birth (Ref:				1.115		
Singleton)				2412		
Multiple birth	4.34***	3.88-4.87	0.00	4.48***	3.98-5.06	0.00
Birth order (Ref: first born)			$\overline{\mathbf{J}}$			
$2^{nd}-4^{th}$	1.18	0.94-1.47	0.03	1.23	0.97-1.56	0.22
5 <sup>th</sup> +	1.34**	1.06-1.69	0.12	1.33**	1.03-1.71	0.48
Death of preceding child						
(Ref: survived)					> //	
Died	2.66***	2.48-2.86	0.11	2.52***	2.34-2.71	0.03
Mother's education (Ref:				$\sim$		
none/primary)				.05		
Secondary/tertiary	2			0.58***	0.54-0.64	0.00
Mother's age (Ref:<18)	4/ 6	Chengo			/	
18-35 years old		henad	hi V	0.61***	0.57-0.65	0.45
36+ years old		·cnyt	,,,,,	0.64***	0.55-0.74	0.00
Miscarriage (Ref: no)						
Yes				1.23***	1.16-1.30	0.07
Wealth Index (Ref: Low)						
Middle				1.05	0.98-1.13	0.69
High				0.90**	0.83-0.98	0.26
Adults in HH (Ref: mother						
alone)						
1 other adult				0.86**	0.78-0.95	0.18
2+ other adults				0.97	0.87-1.07	0.91
Remoteness of health				0.97	0.07 1.07	0.71
services (Ref: no)						
Yes				1.04	0.97-1.11	0.61
No. of subjects			135,657	1.01	0.97 1.11	124,598
No. of failures			6,469			5,927
Time at risk		15	74,015.8		1 4	45,827.2
$\chi^2$		1,5	2710.73			3,131.12
λ p-value			0.000			0.000
	< 0.05 ar	d * for p < 0				0.000

 Table 4: Cox proportional hazards model for determinants of infant mortality in Nicaragua

\*\*\* for p < 0.001, \*\* for p < 0.05 and \* for p < 0.10

In turn, the new covariates to model 2 report on statistically significant expected results such as: mothers with a secondary or higher education reduced their children risk of dying in 42% compared with other non-educated or primary-educated mothers. While, mothers aged 18-35 years old and those older than 36, reduce the risk of death of their children by 39% and 36% respectively. Furthermore, reporting having a termination or miscarriage before negatively affects the risk of death for the index child, as it increases it in 23% compared to mothers that have not had a miscarriage before.

On the household level, children from high-income households were found to have a 10% lower risk of dying than children born to low-income households. And lastly, children to mother's that have one adult company in the household have a 14% lower risk of infant death than those in households where the mother is the only adult. At the same time, a covariate that was not found statistically significant or had an ambiguous impact reflected on their confidence intervals, was remoteness to health care services; because it was a proxy variable not constructed on actual distance in kilometers but rather scarce reports on births at home or having mentioned that the health care centers or hospital were out of reach in other sections of the DHS.

### 4.2.2. Extended Cox proportional hazards model

As mentioned in the last subsection, the proportional hazard assumption was tested through the Schoenfeld residuals (found in the last column to the right of Table 4). For the first model, two covariates were found to be time-dependent, those being: sex of index child and multiple birth. The Cox PH model was extended through the inclusion of the interactions terms between the variables that violate the PH assumption and the logarithm of time, results presented in Table 5. When including the aforementioned interactions, both variables for the first model turned out to be time-dependent, thus, violate the proportionality assumption for that specific predictor.

This means that for these predictors the hazard ratios are not constant over time and assuming a time distribution better specifies the model. For instance, once using the extended Cox PH model for sex of the index child, the effect on the risk of death goes back to 1, which might indicate it not having an impact on the hazard rate of Nicaraguan infants. For the case of single or multiple births, the hazard rate goes down to 0.87 (13% lower risk of dying that single-birth children over time), which evidences that there might be a time frame where the hazard is high for children born from a multiple births but this effect is not proportional on time.

		(1)			(2)	
	HR	95% CI	p-value	HR	95% CI	p-value
IBI (Ref: first born)						
<18	1.36**	1.08-1.70	0.01	1.41***	1.12-1.79	0.00
18-35	0.62***	0.50-0.78	0.00	0.66***	0.52-0.84	0.00
36+	0.37***	0.30-0.47	0.00	0.44***	0.34-0.56	0.00
Sex of index child (Ref:						
male)						
Female	0.80***	0.76-0.84	0.00	0.80***	0.76-0.85	0.00
Singleton birth (Ref:						
Singleton)						
Multiple birth	4.26***	3.80-4.79	0.00	4.42***	3.91-4.99	0.00
Birth order (Ref: first born)						
$2^{nd}-4^{th}$	1.16	0.92-1.45	0.20	1.21	0.96-1.54	0.11
5 <sup>th</sup> +	1.31**	1.03-1.66	0.02	1.31**	1.02-1.69	0.03
Death of preceding child						
(Ref: survived)		TA	2			
Died	2.65***	2.47-2.85	0.00	2.51***	2.33-2.71	0.00
Mother's education (Ref:	X	*		$\times$ $\setminus$		
none/primary)				O. FOrkstuk	0.54.0.54	0.00
Secondary/tertiary				0.59***	0.54-0.64	0.00
Mother's age (Ref:<18)				0.61***	0.57.0.65	0.00
18-35 years old	1			0.64***	0.57-0.65	0.00
36+ years old		Г/т г	-//-	0.64	0.55-0.74	0.00
Miscarriage (Ref: no) Yes			$\overline{\mathbf{J}}$	1.23***	1.16-1.30	0.00
Wealth Index (Ref: Low)				1.25***	1.10-1.50	0.00
Middle			- / III	1.05	0.98-1.13	0.16
High				0.90**	0.83-0.98	0.10
Adults in HH (Ref: mother	-			0.90	0.85-0.98	0.01
alone)	ي ا			ノーた	· //	
1 other adult	2			0.86**	0.78-0.96	0.01
2+ other adults	0			0.97	0.87-1.07	0.55
Remoteness of health services	221					
(Ref: no)	9/	<u>.</u>	1	$o' \land$		
Yes		hona	hi V	1.04	0.97-1.11	0.27
Time varying covariates	HR	95% CI	p-value	HR	95% CI	p-value
Sex of index child x Ln T	1.05***	1.02-1.08	0.00	1.05	1.02-1.08	0.00
Singleton birth x Ln T	0.87***	0.83-0.91	0.00	0.89	0.84-0.93	0.00
Mother's educ. x Ln T	0.87	0.05 0.91	0.00		0.84-0.91	0.00
No. of subjects			135,657	0.88	0.0+-0.71	124,598
No. of failures						5,927
Time at risk		1	6,469 ,574,015.8		1	,445,827.2
$\chi^2$		1	2,761.71		1	3,212.53
χ p-value			0.000			0.000

 Table 5: Extended Cox proportional hazard model for time-dependent variables results for determinants of infant mortality in Nicaragua

\*\*\* for p < 0.001, \*\* for p < 0.05 and \* for p < 0.10

For the second model, also three covariates were found to be time-dependent, those being: sex of index child, multiple birth and mother's education. In this case the interactions between these covariates and time reveal that, again, all three variables for this model are time-dependent and do not comply with the proportionality assumption for each predictor. Regarding the analysis of mother's education, the hazard rate goes down to 0.88 (12% lower risk of dying that those born to non-educated or primarily schooled mothers over time), which evidences that a possible time frame where the hazard is high for children born from low-educated mothers but this effect is not proportional on time.

For this model the interactions of these covariates with the logarithm of time, all variables but death of the preceding child are time-dependent and violate the PH assumption for that specific predictor. Under the premise of consistent findings of time-dependent variables a parametric approach follows in the next subsection, which assumes a time distribution that might help reduce the potential bias on the estimators found for some predictors in the extended Cox PH model.

#### 4.3. Parametric model findings

#### 4.3.1. Weibull distribution model

Parametric survival models assume that the survival time follows a known distribution. For this study, the Weibull distribution model is used because of its great flexibility and adaptability to various time distributions. The results for the Weibull distribution model are presented in Table 6.

As seen by the negative value of p, the shape parameter, the distribution evidenced a hazard that decreases over time. Generally, for both specifications, p does not vary over models, i.e. p goes from -0.81 in the first model to -0.80 if the second model. Thus, the accelerated failure time assumption (AFT) and the proportional hazard (PH) assumption both hold.

The results of the Weibull distribution model are very consistent with those found in the Cox PH model and the extended Cox PH model specifications. In the second model, when considering child, mother and household specific variables, children born in the shortest interbirth interval (<18 months) have a 39% higher risk of death; those born to the 18-35 months interbirth interval have a 35% lower risk, as opposed to the recommended interval that effectively has a lower risk of infant death of 57%. This is consistent with the WHO recommendation of waiting at least 24 months between subsequent birth to pregnancy; i.e. 33 months between birth to birth.

Additional covariates find similar effects to those found in the models depicted in Section 4.2. For instance, female children have a 19% lower risk of dying that their male counterparts; a multiple birth child has 355% higher risk of death than a single one; and the death of the preceding child increases the risk of death 154% when compared to those who didn't have their sibling to die. Conversely, at the mother level secondary or higher

educated mother reduced their children risk of dying by 42% compared to non or low educated mother; and older mothers (18-35 and older than 36 years old) give birth to children with a lower risk of death of 39% and 37%, respectively; finally, those that had had a miscarriage expose their children to a 23% higher risk of death as infants of mothers who didn't have a previous miscarriage.

At the household level, economic resources and family company/support also reported to be influential. Children born to high level income households have a 10% lower risk of death; while mothers accompanied by one adult reduced their children death risk in 14% when compared with mother's that do not have any company/support.

Table 6: Weibull distribution model results for determinants of infant mortality inNicaragua

/		(1)			(2)	
	HR	95% CI	p-value	HR	95% CI	p-value
IBI (Ref: first born)	. Vr					
<18	1.34**	1.07-1.68	0.01	1.39**	1.10-1.76	0.01
18-35	0.61***	0.49-0.77	0.00	0.65***	0.51-0.82	0.00
36+	0.37***	0.29-0.46	0.00	0.43***	0.34-0.55	0.00
Sex of index child (Ref: male)	old I	0.29 0.10	0.00	115	0.0 . 0.000	0.00
Female	0.81***	0.77-0.85	0.00	0.81***	0.77-0.85	0.00
Singleton birth (Ref: Singleton)	0.01		0.00	0.01	0.77 0.05	0.00
Multiple birth	4.40***	3.93-4.93	0.00	4.55***	4.03-5.13	0.00
Birth order (Ref: first born)	1.10	5.75 1.75	0.00	1.55	1.05 5.15	0.00
2 <sup>nd</sup> _4 <sup>th</sup>	0.85	0.68-1.06	0.15	1.23*	0.97-1.56	0.08
5 <sup>th</sup> +	1.14***	1.07-1.21	0.00	1.34**	1.04-1.72	0.02
Death of preceding child (Ref:		1.0, 1.21	0.00	1.51	1.01 1.72	0.02
survived)				_	5 //	
Died	2 68***	2.49-2.88	0.00	2.54***	2.35-2.73	0.00
Mother's education (Ref:	2.68*** 9/ Che	2.47 2.00	0.00	2.34 0	2.55 2.15	0.00
none/primary)				5		
Secondary/tertiary				0.58***	0.54-0.63	0.00
Mother's age (Ref:<18)	91			0.50	0.54 0.05	0.00
18-35 years old			19	0.61***	0.57-0.65	0.00
36+ years old	Cha	nach	jU	0.63***	0.55-0.74	0.00
Miscarriage (Ref: no)		ngun	1	0.05	0.55-0.74	0.00
Yes				1.23***	1.16-1.30	0.00
Wealth Index (Ref: Low)			//	1.25	1.10-1.50	0.00
Middle				1.05	0.98-1.13	0.16
High				0.90**	0.83-0.98	0.10
Adults in HH (Ref: mother alone)				0.70	0.05-0.70	0.01
1 other adult				0.86***	0.78-0.95	0.00
2+ other adults				0.80	0.87-1.07	0.52
Remoteness of health services (Ref:				0.97	0.87-1.07	0.52
no)						
Yes				1.04	0.97-1.11	0.28
	0.02***	0.02-0.03	0.00	0.03***	0.02-0.03	0.28
<u>constant</u>	-0.81***	-0.83, -0.78	0.00	-0.80***	-0.83, -0.78	0.00
<u>ln_p</u>	0.45	0.44-0.46	0.00			0.00
<i>p</i>				0.45	0.44-0.46	
<u>l/p</u>	2.24	2.19-2.30	105 657	2.23	2.18-2.29	104 500
No. of subjects			135,657			124,598
No. of failures		1	6,469			5,927
Time at risk		,	74,015.8			45,827.2
$\chi^2$			2730.51			3,152.74
p-value			0.000			0.000

\*\*\* for p < 0.001, \*\* for p < 0.05 and \* for p < 0.10

Alongside the results for the Weibull distribution model in terms of the hazard, the average marginal effects (AME) for the estimation were also found and presented in Table 7. On an individual level, relative to the one percent, the marginal effects report considerable impacts on the overall probability of dying as an infant as a result of the variables of interest. Additionally, the AME help providing means to compare between groups on the probability of the dependent variable, infant mortality.

	(1)	(2)
	dy/dx	dy/dx
IBI (Ref: first born)		
<18	0.00041***	0.00048***
18-35	-0.00027**	-0.00025**
36+	-0.00034***	-0.00033***
Sex of index child (Ref: male)	X	
Female	-0.00016***	-0.00019***
Singleton birth (Ref: Singleton)	$\sim$ $\times$	
Multiple birth	0.00114***	0.00134***
Birth order (Ref: first born)		
2 <sup>nd</sup> -4 <sup>th</sup>	0.00010*	0.00015**
5 <sup>th</sup> +	0.00021**	0.00023**
Death of preceding child (Ref: survived)		
Died	0.00076***	0.00082***
Mother's education (Ref: none/primary)		
Secondary/tertiary		-0.00048***
Mother's age (Ref:<18)		
18-35 years old		-0.00062***
36+ years old		-0.00059***
Miscarriage (Ref: no)		5 //
Yes		0.00018***
Wealth Index (Ref: Low)		
Middle	101	0.00005
High	bi Ul'	-0.00008**
High Adults in HH (Ref: mother alone)	CUI	
1 other adult		-0.00014**
2+ other adults		-0.00004
Remoteness of health services (Ref: no)		
Yes		0.00003

Table 7: Average marginal effects for Weibull distribution model of determinants
of infant mortality in Nicaragua

\*\*\* for p < 0.001, \*\* for p < 0.05 and \* for p < 0.10

For the case of the birth spacing distribution only accounting for child-specific variables (model 1), having a child on the shortest interval (less than 18 months) increases the probability of infant death in 4.1% in comparison with the first born, while the 18-35 months intervals and the greater than 36 months intervals reduce it in 2.7% and 3.4% respectively. More interestingly, looking at movements between categories, i.e. mothers that decide to space their children not less than 18 months, but space the childbirth in 18-35 months reduce the probability of infant death in 6.8% as a results of the cumulative

effects, while those that decide to wait even further up to 36 months and more may reduce their children probability of infant death in around 7.5% due to the cumulative effects.

Meanwhile, the analysis in model 2, which includes child and mother-specific as well as household variables, shows that having a child on the shortest interval (less than 18 months) increases the probability of infant death in 4.8% in comparison with the first born, while the 18-35 months intervals and the greater than 36 months intervals reduce it in 2.5% and 3.3% respectively. Besides, the effects of movements between birth lengths shows slightly higher effects: mothers that decide to space their child not less than 18 months, but 18-35 months reduce in 7.3% the probability of their children to suffer an infant death, while those that decide to wait even further up to 36 months and more may reduce their children probability of infant death in around 8.1%.

# 4.3.2. Weibull model: By-groups definitions on predominance of causal mechanisms

The final stage of this discussion comprises the empirical exploration of the presence and predominance of the causal mechanisms by which a short interbirth interval might have deleterious outcomes that has previously proven plausible in the models described this far. In past sections, it was discussed that the maternal depletion syndrome might be evidenced through the consistent finding of a negative impact of the shortest intervals in the survival of infants, as well as in the deleterious impact of having its previous sibling die for the index child. The second causal mechanism discussed in the light of the previous findings is sibling competition. Mostly reflected on the how the birth order might alter the household's resources allocation and the negative impact of multiple births as infants of the same ages compete for resources and care. The Weibull distribution model was ran in by-groups specifications and the results are presented in Table 8.

For the exploration of the presence and magnitude of impact of the maternal depletion mechanism, the Weibull model was used but dividing the sample in groups of interest. In this regard, not only the shortest IBI was found to have a negative impact, but also how young a mother is at childbirth, the latter which might be the case of an unexperienced and less knowledgeable mother, besides her physiological unreadiness for childbearing and childbirth due her young age. Thus, the group divisions will help explore whether the effects found seem to be more inclined towards a case of mother depletion syndrome, through running model 2 for categories of the death of the preceding child; or to a 'physiological unreadiness' effect, through model 2 also run in the groups of underaged mothers (<18 years old) and older mothers ( $\geq$ 18 years old).

	Death of chi	Death of preceding child	Mother's :	er's age at birth	Index c	Index child's birth order		Wealth index	X	Area of	Area of residence
	Child survived	Child died	<18 years old	≥18 years old	2 <sup>nd</sup> -4 <sup>th</sup>	5 <sup>th</sup> +	Low income	Middle income	High income	Urban	Rural
IBI (Ref: first born)			•								
<18	$1.62^{***}$	1.02	2.07***	$1.36^{**}$	Ref: IBI<18	Ref: IBI<18	$1.61^{***}$	$1.72^{**}$	0.71	1.09	$1.62^{**}$
18-35	$0.72^{**}$	$0.54^{***}$		0.62***	$0.60^{***}$	0.46***	0.74*	0.76	$0.39^{***}$	$0.51^{***}$	0.75*
36+	$0.49^{***}$	$0.32^{***}$	0.55	0.42***	$0.37^{***}$	$0.34^{***}$	$0.51^{***}$	$0.48^{**}$	$0.22^{***}$	$0.33^{***}$	$0.50^{***}$
Sex of index child (Ref:			6			>	/				
Lemale Female	$0.82^{***}$	$0.78^{***}$	0.76***	$0.83^{***}$	0.83***	$0.84^{***}$	0.83***	$0.80^{***}$	$0.74^{***}$	$0.81^{***}$	$0.81^{***}$
Singleton birth (Ref:			$C_{f}$				Ţ				
Singleton) Muttin Lint	1 1 4 * *	*** 77	***	***00	101***	***07 7	×**	A 00 ***	*** ? 0 U	0 <u>4</u> 0**	и ОЛ***
Multiple official Birth order (Ref: first born)	4.14		1.04 N	4.23	4.21		4.09				
$2^{nd}$ - $4^{th}$	1.12	0.82	0.77	1.33**			1.00	1.06	2.88***	$1.77^{***}$	0.97
$5^{th}+$	1.16	1.01	1.48	$1.44^{**}$			1.11	1.01	$3.13^{***}$	$1.93^{***}$	1.05
Death of preceding child			ni				44				
(Ref: survived) Died			***LC C	2 59***	236***	0 95***	***92 0	×**CC	2 13***	2 29***	2 70***
Mother's education (Ref:			, n					i	ì	Ì	
none/primary)				4							
Secondary/tertiary Mother's age at birth	0.59***	$0.52^{***}$	0.56***	0.60***	0.59***	16.0	0.65***	$0.61^{***}$	0.52***	0.58***	0.60***
18-35 years old	$0.60^{***}$	$0.65^{***}$		5	$0.65^{***}$	0.42*	0.63***	$0.64^{***}$	$0.51^{***}$	$0.60^{***}$	$0.62^{***}$
36+ years old	$0.57^{***}$	1.04	/		$0.43^{***}$	0.46	$0.70^{***}$	$0.40^{***}$	$0.57^{**}$	$0.67^{***}$	$0.62^{***}$
Miscarriage (Ref: No)											
Vec	1 27***	1 04	1 51***	1 1 1 * * *	106***	1 03	1 しん***	1 18**	1 1 8 * *	1 74**	***oo -

	Death of ch	Death of preceding child	Mother's	age at birth	Index child's birth order	d's birth er		Wealth index		Area of r	Area of residence
	Child survived	Child died	<18 years old	≥18 years old	2 <sup>nd</sup> -4 <sup>th</sup>	5 <sup>th</sup> +	Low income	Middle income	High income	Urban	Rural
Wealth Index (Ref:											
Low) Middle	1.03	1.15	1.08	1.04 B N	1.12**	0.88				1.01	1.01
High	$0.89^{**}$	0.89	66.0	$0.87^{***}$		0.84*				$0.84^{***}$	1.01
Adults in HH (Ref:			9			T					
1 other adult	$0.89^{**}$	0.75**	1.01	0.81***	$0.84^{**}$	0.74**	0.87***	$0.67^{***}$	1.23	$0.84^{**}$	0.90
2+ other adults	1.02	0.72**	1.04			0.88	0.98	$0.80^{**}$		0.94	1.01
Remoteness of health			96		2		白衣				
services (Ref: no)			'n		E						
Yes	1.07*	0.88	1.08	1.03	1.00	0.98	1.03	$1.27^{**}$	0.80	1.05	1.05
constant	$0.03^{***}$	$0.16^{***}$	$0.02^{***}$	$0.02^{***}$	0.03***	0.08***	0.03***	$0.03^{***}$	$0.02^{***}$	$0.03^{***}$	$0.03^{***}$
$d^-ul$	-0.80***	-0.80***	-0.79***	-0.81***	-0.76***	-0.81***	-0.79***	-0.78***	-0.90***	-0.81***	-0.80***
d	0.45	0.45	0.46	0.45	0.47	0.45	0.45	0.46	0.41	0.45	0.45
d/p	2.23	2.22	2.19	2.25	2.13	2.24	2.20	2.18	2.47	2.24	2.23
No. of subjects	117,830	6,780	I	104,863	61,649	25,366	76,949	21,989	25,660	55,966	68,632
No. of failures	5,009	918	1,493	4,434	2,793	1,365	4,022	1,055	850	2,431	3,496
Time at risk	1,372,382	73,445.17	224,359.3	1,221,467.87	717,137.4	293,000.9	890,216.6	255,107.2	300,503.4	652,243.5	794,583.63
$\chi^{2}$	1835.46	514.66	508.45	2324.34	1882.44	778.11	1892.5	648.01	505.98	1330.76	1816.73
p-value	0.00	0.00	0.00	0.00	0.00	0.00	0.00	00.00	000	00.00	00.00

Additionally, for the study of the presence and intensity of the impact of sibling competition, again the Weibull model was used in accordance with groups that provide insights on the mechanism. In one hand, sibling competition is expected to be reflected on the higher negative effect in the hazard subject to the short interval of the lower order children; and on the other hand, the household income, since the competition might be fueled by the additional scarcity of resources due to poverty, in which case, well-off households will not suffer as much from this mechanism.

Therefore, this second group divisions will aid on exploring whether the effects found might be more inclined towards sibling competition, by running the model for the categories of birth order; or to a 'wealth-centric' effect, through the separation of the sample by income-level groups. Lastly, a second group division that might help evidence the inequality of resources, or access to them, is the area of residence; under the premise than families living in rural areas would have less access to proper and constant health care making complications that originate from the short interbirth interval more risky for their offspring.

For the case of assessing the predominant impact of the causal mechanism maternal depletion syndrome or the empirically proposed 'physiological unreadiness' effect, when dividing groups where the preceding child survived or died, it would be expected to find a greater hazard for those mothers that lost their previous child and shortly spaced the next, not giving herself enough time to replenish the nutritional stores required to pursue another pregnancy and childbirth. Nonetheless, for the case of index children that had their sibling die the shortest interbirth interval has a neutral statistically non-significant effect, while the main deleterious effect of the short interval can be observed in the case were the previous sibling survived. Nonetheless, one effect that can be observed for 'depleted' mothers (those that had lost their previous child) is the higher risk the index child is placed on due to being born to a multiple birth. Thus, indicating that there might be mothers that are already depleted as a results of their previous lost and have a next child with a higher risk when it's a multiple birth possibly due to her unrestored nutritional stores to bear a healthy child.

Nevertheless, the 'physiological unreadiness' effect can be more clearly seen in the group division by mother's age. Where teenage mothers are found to have a clearly larger deleterious effect of the shortest interval, having these children more than 107% higher risk of death, in contrast with a 36% higher risk of death of their counterparts born to adult mothers. In this sense, the main risk factors of teenage mothers revolve around physiological unreadiness and distress to their bodies since variables related to household wealth or adult company are not statistically significant.

For young girls that experienced menarche, it might indicate that they are biologically able to conceive but that does not translate into being able to reproduce, or carry the pregnancy to full-term (Montagu, 1981). Young mothers are, in a sense, competing for nutrients and growth with their own children; and childbearing and childbirth put them at greater risk of complications both for her own survival and for the health outcomes of her children (WHO, 2006).

Thus, under the assessment of which effect prevails with the selected and available data, the Nicaraguan experience proves to have a greater issue: the increasing number of teenage mothers, their unreadiness for childbirth both physically and psychologically, and the pressing hazard associated with them having shortly spaced children when compared to adult mother and longer child spacing.

In second place, when exploring to decompose the short interbirth interval deleterious effect, there's also the possibility of it containing a share due to the competition among closely aged children in the same family. Additionally, variables such as the birth order might also help to shed light into the sibling competition effect since older siblings might get a preferential treatment or might hoard resources. The findings for the models divided by these groups is unable to evidence it affecting the hazard for children as a result of the short interval. This is because the first-born group is not a subject to the calculation of the IBI and for all specifications it would exclude the reference category used in all models, making them unable to compare themselves.

Regarding the wealth and accessibility to health care services group that might reflect on a struggle for resources in general and boost sibling competition, low- and middle-income households are found to have children with a higher risk of death associated with spacing them with the shortest interbirth interval. Children born to a low-income household in a lesser than 18 months interval have a higher risk of death that their first-born counterparts of 61%, 72% for children in the same situation but born to middle income households and 62% to those born in the shortest interval but in rural households.

Conversely, for high-income families and those in urban areas the shortest interval effect is found to be neutral and/or statistically non-significant. Wealthier mothers and those living in urban areas can either be readily available to purchase private health care services or have an easier anytime access to State-provided health care. Further study and more comprehensive variables would be of great help to properly assess the actual

presence of a sibling competition mechanism and whether the 'wealth-centric' effect predominates as the main mechanism of the two compared.

Lastly, a secondary exploration using the Weibull model was conducted for sibling competition that might arise as a result of the gender of the child (See Table 10 in Appendix), using for reference, the clear preferences other societies have for male offspring leaving girls unattended and at higher risk of death. Nonetheless, for the case of Nicaragua, there's no clear evidence that there exist clear differences in the hazard of death as a result of the gender distributions. For instance, the hazard associated with the shortest interval represents a similar hazard for both, male and female children, as it has a 37% higher risk for male children compared to the first born; and a 43% higher risk for female children when compared to the first born. Similarly, the results for other variables that might indicate the presence of sibling competition don't present significant differences among groups. Thus, there's no evidence that gender related reasons play a decisive role for the survival of either groups, nor it validates the hypothesis of gender preferences among parents seen in other cultures and societies.

## **5** Limitations of the study

One of the biggest limitations of this study is regarding the data source, namely the use of pooled data from the Demographic Health Survey (DHS). Mainly, it's important to address limitations related to: (1) the type of data collected; and (2) the sampling definitions in the DHS.

Regarding the type of data collected, as it's pointed out by Hobcraft, et al. (1983), the DHS relies on retrospective birth-history reported by the mothers themselves, which is subject to quality issues such as omission and misplacement. The misplacement of births or misreports of age of death are great challenges for self-reported data, particularly for children that died, that are not living with their natural mother or that were born a long time ago from the date of the survey (Ahmad, et al., 2000). Those misreports can ultimately cause overestimations of children born in the short interval (Hobcraft, et al., 1983). The second issue, omission, is considered the most serious one, as omitting the birth and death of a child all together can artificially enlarge the intervals, thus, biasing downwards the potential effect of the shortest intervals (Hobcraft, et al., 1983).

On the other hand, there are other issues arising from the sampling definitions used to limit the target subjects of the DHS. In this sense, since the main sampling unit for reproductive history is women of reproductive ages (15-49 years old), those mothers that have passed away –for reasons that can potentially be related to the length of the birth interval and its deleterious effect– and her children are omitted from the dataset; biasing child mortality downwards (Ahmad, et al., 2000). Additionally, the sampling definitions for children places challenges for estimations with the DHS: only livebirths are taken into consideration for the full self-reported birth record, children that died preterm (miscarriages and stillbirths) are not considered and the lack of information on these deaths can be masking bigger deleterious effects of birth spacing; secondly, relevant information on live outcomes such as breastfeeding practices, health service utilization, anthropometry, and morbidity, were only collected for children alive, living at the household and younger than five years of age. Thus, not providing the full picture for all the offspring detailed in the birth records and limiting on important covariates contained in that information (Boerma & Bicego, 1992).

Despite the limitations described above, the DHS still represent for developing countries –where data availability is limited– the best sources of information on reproductive behavior (Ahmad, et al., 2000). When comparing it to hospital records, Rutstein (2005) advocates for the DHS data on developing countries, since they have low rates of institutional births, and these data source also provide more information past the neonatal period and account for a larger and more comprehensive set of covariates.

Lastly, one big limitation for the mechanisms assessment part of this study is the lack of medical records on important information regarding the mother's health status. Only by knowing detailed information on a mother's nutrient stores during her pregnancies is that a proper evaluation of the maternal depletion syndrome mechanism can be evidenced as well as the detailed pathways by which a teenage mother is more likely to have more complications during childbearing and childbirth.

#### **6** Conclusions

Child mortality is world-level health tragedy that prevented around 5.3 million children in 2018 from reaching five years of age, and even more alarming is the fact that 75% of those children didn't even lived past their first year. Despite Latin America being one of the regions that led the reduction of child mortality rates from the 1980s to the 2000s, as a country Nicaragua has still plenty of work to do. Infant mortality in the country has continued to decline but at a very slow rate (0.6% to 1%) and maintaining the position of the second largest infant mortality rate in Central America with 15.7 deaths per 1,000 live births.

Among the actions to be considered to reduce the risk of adverse maternal, perinatal and infant outcomes, the World Health Organization (WHO) suggests waiting at least 24 months after a live birth to attempt another pregnancy, that is, an interbirth interval of 33 months or roughly 3 years. This is also extensively backed up on international multidisciplinary research that has found short birth spacing to have deleterious effect on child and maternal wellbeing and survival.

Nicaragua's government has stated in the national health strategy plans that child spacing is a policy tool that should be addressed when aiming to protect mothers and children lives. Nonetheless, the issue lies in the lack of objective policy actions proposed nor taken under this premise and more prominently, the lack of evidence that such relationship does in fact exists and which mechanisms does it work through for the case of Nicaragua.

This study used survival analysis on the pooled data from the Nicaraguan DHS 1998-2011, to provide evidence on the association of child spacing along and infant mortality and shed light on which mechanism these results will mostly be associated with. On the non-parametric approach, the Kaplan Meier curves for the survival probability in accordance to the length of the interbirth intervals showed that the lower survival probability, and clearly separated from other categories, belongs to the children born in the shortest birth interval (less than 18 months); while children born from the WHO recommended interval (more than 36 months) had the highest survival probability curve.

The Cox proportional hazard model, the extended Cox PH model and the Weibull distribution model all provided consistent parameters for the main dependent variable, the interbirth interval, and the set of confounding factors. The Weibull distribution model is preferred since it assumes a shape for the survival time distribution overcoming the issue of time-dependency of variables that prevails in the Cox PH model.

Children born in the shortest interbirth interval (<18 months) have a 39% higher risk of death; while those born to the recommended interval that effectively has a lower risk of infant death of 57%. These results are consistent with the WHO recommendation of waiting at least 33 months between births. This effect summed to the negative effect of the death of the preceding child provided initial information to suggest that mothers might potentially suffer from maternal depletion syndrome.

The second mechanism that the results indicate is the sibling competition mechanism, in which older siblings may dominate most of the resources leaving youngerorder siblings with limited access to those household resources; or having a same-aged sibling born at the exact same time means having a direct rival for the same kind of resources from the time of birth, e.g. mother's care and attention, breastfeeding time and quality, particularly seen in the greater hazard for children of the 5<sup>th</sup> order or higher and those born from multiple births.

Using an exploratory empirical approach, the predominance of one mechanism over another was assessed alongside effects seen in the reality of Nicaraguan families. Firstly, maternal depletion syndrome cannot be clearly evidenced from the case were the previous sibling died in its effects on the interbirth interval but it is found to have an effect for children that had their previous sibling die and are born to a multiple birth. Additionally, the 'physiological unreadiness' effect can be clearly seen in the group division by mother's age. In this sense, teenage mothers are found to have a clearly larger deleterious effect of the shortest interval, having these children more than 107% higher risk of death, in contrast with a 36% higher risk of death of their counterparts born to adult mothers. The Nicaraguan experience proves to have a greater issue regarding interbirth intervals: the increasing number of teenage mothers, their unreadiness for childbirth both physically and psychologically, and the pressing hazard associated with them having shortly spaced children when compared to adult mother and longer child spacing.

Regarding the sibling competition, the group defining variable used an effect of the shortest interval could not be produced in a way that's comparable with other findings since birth order was to be used. Nonetheless, for the 'wealth-centric' effect, children born to a low-income household in a lesser than 18 months interval have a higher risk of death that their first-born counterparts of 61%, 72% for children in the same situation but born to middle income households and 62% to those born in the shortest interval but in rural households. Thus, wealth and accessibility to health care services does reflect more clearly the struggle for resources and boost any form of 'sibling competition', while, wealthier mothers and those living in urban areas can either be readily available to purchase private health care services or have an easier-anytime access to State-provided health care that can help them better tackle potential deleterious effect on the shortest interval in their offspring health outcomes.

Future studies on the light of more comprehensive variables would be of great help to properly assess the actual presence of either mechanism, maternal depletion syndrome and sibling competition mechanism; for instance, conducting a quasiexperiment on nutritional store values for mother's at a certain stage of pregnancy for those who spaced their children closely or to the recommended interval. Additionally, it would be contribution to the discussion, addressing a quite possibly large impactful mechanism of a short interbirth interval on infant death: early weaning and lower quality feeding and care for children so closely spaced; only explorable through information on the breastfeeding practice of the mothers.

The results presented in this study do find evidence on the deleterious effect an interval of less than 18 months for the survival of infants, nonetheless, the pathways through which this effect operates should be further discussed was there more complete data on a Nicaraguan mothers reproductive and sexual health practices and status. It's found that there's a larger deleterious more predominant effect prevails for teenage mothers that space their children closely, possibly due to her unreadiness for childbearing and childbirth. It's also found that the wealth of the household and the accessibility to public health care of which urban areas are benefited from can play a large role in diminishing the effect of the deleterious effect of the short birth interval. A higher income can also be associated with more abundant resources, such as food and nutritional complements that might as well lessen the hazard on shortly spaced children.

Based on these results, public policies should aim to generate quality information to properly address the channels that explain the deleterious effect of the intervals of less than 18 months. In this sense, since the government's strategy plans already recognize this WHO recommendation and its importance, it's crucial to translate those statements into policy actions, especially those aimed to the youth, such as objective sex education and easy-broad access to contraceptives.

Only suggesting educating mothers on the importance of them spacing their children with an adequate timing would not be enough to subdue theses effects on infant mortality. In turn, the concrete policy implications in the light of these results should be segmented by age groups and respond to different capabilities of family responses to deal with the potential deleterious effects of a short-spaced pregnancy and childbirth. Particularly, for the case of the larger deleterious effect of short birth intervals on teenage mothers and their offspring, only the combination of a more comprehensive sex education that reaches not only the youth in schools but also those that drop out of the system or those that have never been part of it, and that covers both contraceptive uses and healthy sexual practices (including proper spacing); in hand with the access to those means of protection, counseling, and health care through local health care units. For mother's depletion syndrome in young and adult mothers alike, the challenge should also address the improvements of the conditions for the poorest mothers and those that have constrained access to health care. In particular, the health care system must not only improve more their outreach to isolated communities, but also provide free and complete nutritional supplements to mothers that have a close child spacing.

Furthermore, for sibling competition boosted through a wealth centric effect: considering the vulnerability that mothers from low-income households have towards accessing and purchasing means to subdue the effects of the shortest intervals, the government can implement maternal subsidies, for low-income mothers and especially those in rural areas, for current pregnancies shortly spaced with its preceding childbirth. Nonetheless, it would be key to allocate these subsidies in a way so that they don't incentivize shortly spaced births, but rather, a maternal subsidy that's a conditional cash transfer on the basis of participating of sex and reproductive education programs, family planning, and spacing their children with a minimum of 18 months. These segmented policies will bear the best results in the challenge of reducing infant mortality and increasing Nicaraguan families' wellbeing in the years to come.



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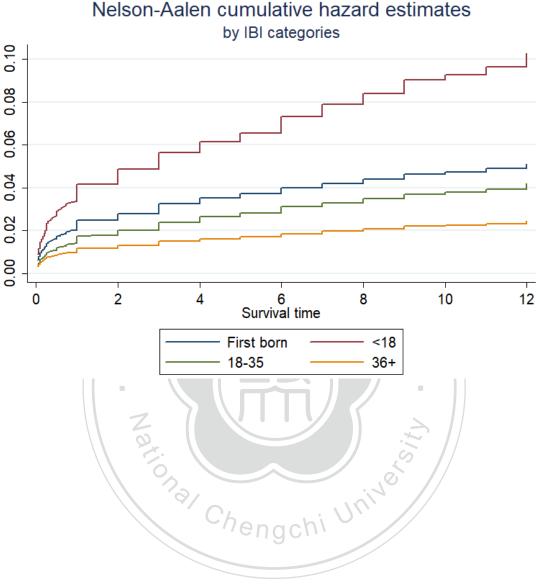
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# Appendix



**Figure 2: Nelson Aalen cumulative hazard for Interbirth intervals** Nelson-Aalen cumulative hazard estimates

		(1)			(2)	
	Coef.	95% CI	p-value	Coef.	95% CI	p-value
IBI	-0.001***	-0.001, -0.001	0.000	-0.0004***	-0.0005, -0.0004	0.000
Sex of index child (Ref: male)						
Female	-0.009***	-0.012, -0.007	0.000	-0.0092***	-0.0121, -0.0063	0.000
Singleton birth (Ref: Singleton)						
Multiple birth	0.154***	0.142, 0.165	0.000	0.1483***	0.1365, 0.1601	0.000
Birth order (Ref: first born)	0.002***	0.001, 0.003	0.000	0.0049***	0.0039, 0.0060	0.000
Death of preceding child (Ref:						
survived)						
Died	0.085***	0.080, 0.090	0.000	0.0837***	0.0782, 0.0891	0.000
Mother's education (Ref:						
none/primary)						
Secondary/tertiary				-0.0131***	-0.0173, -0.0088	0.000
Mother's age				-0.0020***	-0.0024, -0.0016	0.000
Miscarriage (Ref: no)						
Yes				0.0072***	0.0038, 0.0106	0.000
Wealth Index (Ref: Low)						
Middle				0.0038*	-0.0003, 0.0079	0.068
High		7/1 ;	1.	0.0030	-0.0015, 0.0075	0.185
Number of adults in HH		IEX /		-0.0001	-0.0012, 0.0010	0.884
Remoteness of health services (Ref:		V/				
no)				0.0000	0.00.00.00011	0 1 5 4
Yes	0.000	0.050 0.055	0.000	-0.0029	-0.0069, 0.0011	0.154
constant	0.062***	0.058, 0.066	0.000	0.0951***	0.0869, 0.1034	0.000
No. of observations			94559			87029
Adjusted R <sup>2</sup>	7		0.0265	Adim		0.0282
Prob > F			0.000			0.000
*** for p < 0.001, ** f	for $p < 0.05$	and $*$ for p <	0.10			
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## Table 9: Linear probability model for infant mortality in Nicaragua

		Male			Female		
	HR	95% CI	p-value	HR	95% CI	p-value	
IBI (Ref: first born)							
<18	1.37**	0.99-1.89	0.05	1.43**	1.01-2.02	0.05	
18-35	0.64**	0.46-0.88	0.05	0.67**	0.47-0.95	0.03	
36+	0.41***	0.29-0.57	0.00	0.45***	0.32-0.65	0.00	
Sex of index child (Ref: male)	0.11	0.29 0.37	0.00	0.15	0.52 0.05	0.00	
Female							
Singleton birth (Ref: Singleton)							
Multiple birth	4.47***	3.79-5.27	0.00	4.65*	3.90-5.54	0.00	
Birth order (Ref: first born)	,	5.17 5.21	0.00	1.05	5.90 5.51	0.00	
2 <sup>nd</sup> -4 <sup>th</sup>	1.22	0.88-1.68	0.23	1.24	0.87-1.77	0.23	
5 <sup>th</sup> +	1.34*	0.95-1.89	0.09	1.32	0.91-1.92	0.14	
Death of preceding child (Ref:	110 1	0170 1107	0.07	1.02	0001 1002	0.1	
survived)							
Died	2.62***	2.37-2.90	0.00	2.42***	2.16-2.71	0.0	
Mother's education (Ref:							
none/primary)							
Secondary/tertiary	0.60***	0.54-0.67	0.00	0.56***	0.50-0.64	0.00	
Mother's age (Ref:<18)		1 .1					
18-35 years old	0.60***	0.55-0.66	0.00	0.62***	0.56-0.69	0.0	
36+ years old	0.61***	0.50-0.75	0.00	0.66***	0.53-0.83	0.0	
Miscarriage (Ref: no)			9	$\times$	\		
Yes	1.16***	1.07-1.25	0.00	1.32***	1.21-1.44	0.00	
Wealth Index (Ref: Low)							
Middle	1.07	0.97-1.17	0.16	1.03	0.93-1.15	0.57	
High	0.94	0.84-1.05	0.25	0.85**	0.75-0.96	0.0	
Adults in HH (Ref: mother alone)				1-1-			
1 other adult	0.86**	0.75-0.99	0.03	1.05	0.95-1.17	0.30	
2+ other adults	0.93	0.81-1.07	0.30	0.86**	0.73-1.00	0.05	
Remoteness of health services (Ref:							
no)							
Yes	1.03	0.94-1.12	0.59	1.02	0.87-1.19	0.85	
constant	0.03***	0.03-0.04	0.00	0.02***	0.02-0.02	0.00	
ln_p	-0.84***	-0.87, -0.80	0.00	-0.76***	-0.80, -0.72	0.00	
p	0.43	0.42-0.45		0.47	0.45-0.49		
l/p	2.31	2.23-2.39		2.13	2.05-2.22		
No. of subjects			64367			60231	
No. of failures	91		3349		/	2578	
Time at risk	1 CL	74	3738.33	× _//	70	02088.83	
$\chi^2$	al Ch	enach	1736.42			1371.5	
p-value		<u>-igo</u> i	0.000			0.000	

## Table 10: Weibull distribution model results by gender of Nicaraguan children

\*\*\* for p < 0.001, \*\* for p < 0.05 and \* for p < 0.10