

Impact of Child Health and Disability on Subsequent Maternal Fertility

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Abstract: Prevalence of chronic conditions among children has been rising in the past four decades. It is generally estimated that between 2 and 7 percent of children in the US are disabled, depending on the measure of activity limitations used. At the same time, the prevalence of low birth weight (LBW) and preterm birth infants is increasing and larger proportions of LBW and preterm infants are surviving the perinatal period due to improved technology and care, many of whom subsequently experience chronic conditions. Given these trends, it is important to understand the effect of experiencing LBW and preterm births and child disability on subsequent childbearing. Despite the policy relevance, there has been limited investigation into the impacts of LBW, preterm birth and child disability on subsequent reproductive behavior, particularly in the US. In this study, we examine whether giving birth to a LBW, preterm or disabled child impacts subsequent maternal fertility. We empirically estimate a model of fertility behaviors and outcomes and the impact of health problems and disabilities in a mother-level fixed effects framework. Our sample includes a panel of women who have begun to engage in fertility behavior as measured by at least one conception, resulting in a live birth with measurable health/disability. We employ a discrete-time hazard model that studies maternal fertility behavior/outcome over time as a function of previous child health conditions or disabilities, using merged data from the 1993 National Health Interview Survey (NHIS) and 1995 National Survey of Family Growth (NSFG). The fixed-effect model suggests that having a disabled child or a child born LBW or preterm reduces the probability of subsequent conception or live birth by about 0.1-- 0.14, with overall minimal differences in effects among the three health/disability measures. Significantly lower effects are observed using the random-effect model. In conclusion, we find significant effects of child disability, LBW and preterm birth on reducing subsequent maternal fertility. Models ignoring unobserved "fixed" heterogeneity, such as preferences for health, risk taking, and fertility and health risks may significantly underestimate the effects of health shocks on subsequent reproduction.

I. Introduction

The prevalence of chronic conditions and other health limitations among children has been rising in the past four decades (Perrin et al., 2007). While definitions of disability vary widely leading to different point prevalence estimates, it is generally estimated that between 2 and 7 percent of children in the US are disabled based on activity limitations, with the lower estimate being counted by the definition used by the Social Security Administration and the upper estimate being according to the definition used in the National Health Interview Survey (NHIS) (AHRQ, 2003; Perrin et al., 2007). The disability rate increased from less than 2% in 1960 to 7% in 2004 based on the NHIS activity-limitation measure. However, developmental disabilities, which are chronic physical and mental health conditions that occur during childhood and may have significant effects on activity limitations and functioning such as attention deficit and hyperactivity disorder (ADHD), blindness, autism and others are estimated to occur at higher rates. In the 1997-2005 NHIS, about 13.2% of children 3-17 years of age were estimated to have at least one developmental disability (Boyle et al, 2009).

Similarly, the rates of low birth weight (LBW) and preterm birth have been on the rise during the past three decades. Between 1980 and 2007, the population-level rates of LBW increased from 6.8% to 8.2%, respectively, while preterm birth rates increased from 8.9% to 12.7% (Heron et al., 2010; National Center for Health Statistics, 1982). Furthermore, survival rates, including LBW births, have increased over the second half of the 20th century due to improved technology and care (Kaiser et al., 2004; Lee et al., 1995). Between 1995 and 2001, the LBW infant mortality rate decreased by about 9.3% (Arias et al., 2003). However, these children suffer from subsequent chronic health conditions and developmental and learning disabilities (Anderson and Doyle, 2003; Frankel et al., 1996; Gluckman et al., 2008), some of which extend into adulthood (Currie, 2009; Victora et al., 2008). Also, preterm birth and low

birth weight may require several hospital interventions and extend hospital length of stay and raise costs (Almond et al., 2005).

At the same time, the fertility rate in the US, defined by the number of births per woman, has been relatively steady over the past three decades. Specifically the number of births per 1000 women was 68.4, 70.9, 65.9, and 68.6 in 1980, 1990, 2000 and 2008, respectively (Heron et al., 2010; Martin et al., 2010). Given the slight decline in fertility rates after 1990 but rising rates of child disability, it is important from multiple policy perspectives to understand the effect of child disability on subsequent fertility. Despite the policy relevance and a long economic literature on the quantity-quality tradeoff in children, there has been limited investigation into the impacts of early life child disability, LBW or preterm birth on subsequent fertility behavior among mothers in the US.

In spite of the interest in the household behavioral response, particularly maternal labor supply and marital outcomes, to child health and disability, there are only a few studies specifically examining fertility behavior. This lack of attention is interesting because it could be argued that fertility decisions subsequent to the birth of a child with disabilities or health problems such as preterm birth and LBW is related to both marital dissolution and maternal labor supply decisions. Furthermore, the micro-level decision making with regard to fertility subsequent to the birth of a disabled or unhealthy child has potential macroeconomic impacts for subsequent generations of the labor force. Specifically, if women tend to stop fertility due to the birth of an unhealthy or disabled child, and these unhealthy or disabled children survive to adulthood and continue to experience activity limitations, then the proportion of adults in the subsequent generation with limited functioning and health problems is likely to grow.

We are aware of two previous studies that have examined the micro-level fertility behavior of a representative cohort of US women subsequent to giving birth to a child with

disability (MacInnes, 2008; Park et al., 2003). However, these studies estimate effects of child disability on subsequent fertility-related decisions by comparing mothers of disabled and non-disabled children. However, these comparisons may result in biased estimates due to differences in unobserved preferences, maternal health factors and other family-level effects that may vary between these two groups of mothers (Rosenzweig and Wolpin, 1988).

In this paper we revisit the question of the impacts of child disability or poor health on fertility behavior and outcomes, acknowledging the potential heterogeneity in unobserved characteristics that relate to both child disability/health and fertility decisions. We root our conceptual model in the rational choice framework, specifically the quantity-quality tradeoff model found in Becker and Lewis (1973), Becker and Tomes (1976), and Becker (1991). However, our empirical model is unique in using mother fixed effects models that only utilizes within-mother variation and allow us to capture the effect of disabled/unhealthy children on subsequent fertility outcomes net of any time invariant unobserved heterogeneity in preferences and health among mothers. We compare these estimates to random-effect models that use between-mother variation in order to obtain information on the direction and magnitude of the bias in previous studies failing to account for this unobserved heterogeneity.

In addition to dealing with this issue of unobserved heterogeneity, we address other limitations of previous studies of the effect of child disability on subsequent fertility. First, we evaluate the effects of other health problems besides disability including LBW and preterm birth, which are common, costly and burdensome conditions that deserve specific assessment for their potential effects on fertility behavior. Further, using at-birth health measures allows us to examine the issue of the timing of disability realization are a limitation for studies that only evaluate disability measures. Second, we reexamine the effect of disability on fertility more broadly by evaluating multiple subsequent fertility outcomes and behaviors including conception, live birth and sterilization. Finally, we estimate a model that considers how child

disability may affect the timing to next birth, conditional on having a subsequent live-birth, *which yields new insights to* how disability may affect birth spacing among mothers who have an additional child. Our results suggest there is substantial downward bias in effects of child disability and health problems on subsequent fertility decisions in models that fail to account for unobserved heterogeneity in preferences or health of mothers.

II. Literature Review

There is a relatively broad literature on the socioeconomic consequences of poor child health or disability. A large portion of the literature in this area addresses the relationship between child health and marital stability (Corman and Kaestner, 1992; Fertig, 2008; Joesch and Smith, 1997; Mauldon, 1992; Reichman et al., 2004). This literature finds unhealthy children tend to reduce marital stability, particularly in those of lower socioeconomic status. Of interest to policy makers in the US, Fertig (2008) finds there is heterogeneity in this finding between data from the US and the UK, suggesting system-level or social factors may impact the ability of parents to cope with unhealthy children.

The second area of focus in this relationship has been on child health or disability on the labor supply of mothers (Kuhlthau and Perrin, 2001; Porterfield, 2002; Powers, 2001; Wehby and Ohsfeldt, 2007; Wolfe and Hill, 1995). As theory would predict, maternal labor supply is reduced when an unhealthy child is present in the household. However, this effect also interacts with marital status. Specifically when an unmarried mother's unhealthy child reaches school age they are more likely to increase their labor supply, whereas those women who are married and who are more likely have greater access to other sources of economic support tend not to increase their labor supply when their unhealthy children reach school age (Porterfield, 2002). Furthermore, both marital status and race may interact with child disability in affecting labor supply. Specifically, African-American single mothers are most affected

followed by White single and married mothers, but there are no significant effects of African-American married mothers (Wehby and Ohsfeldt, 2007). Another important point highlighted in this literature is that the reporting of disability is endogenous to labor supply due to unobserved preferences (Powers, 2001).¹ These unobserved preferences also likely impact subsequent fertility decisions, and have not been accounted for in previous estimates of the impact of disabled or unhealthy children on subsequent fertility in a US setting.

As mentioned above, there are relatively few studies examining the impact of unhealthy or disabled children in the household on subsequent fertility decisions, despite it being related to marital dissolution and labor supply. In a more general theoretical framework of household resource allocation, Rosenzweig and Wolpin (1988) examined the relationship between child health, measured by weight standardized for age, and an array of behaviors including subsequent fertility. They found a positive impact of family health endowment² on the total number of children ever born in the family and that healthier children (higher normalized birth weight) born during the follow-up period increased the probability of a subsequent birth in a three-year interval (i.e. reduced birth spacing). However, the authors use 1968-1974 data on 223 households from a single Colombian village, and their approach may not generalize to the US or to disability more clearly defined.

More recently, Park, Hogan and Goldscheider (2003) used linked data from the 1993 National Health Interview Survey (NHIS) to the 1995 National Survey of Family Growth (NSFG) to study the impact of child disability on tubal sterilization. The authors found a positive association between having a disabled child and tubal sterilization. Later work by MacInnes (2008) using these same data examined the subsequent childbearing of women whose first

¹ Reporting of child health conditions are also potentially endogenous to labor supply.

² This was based on the residuals of regression of child health on various child health inputs.

child had a disability. While addressing an important question, these studies did not provide full insight into the effects of child disability on fertility.

The first limitation, from a theoretical standpoint, is that unobservable parental preferences for fertility, risk taking, and health that simultaneously impact both child health and disability as well as fertility decisions. For example, reduced risk-aversion may increase conception and reduce sterilization but may also increase risky behaviors such as smoking, sexually transmitted diseases, and others that may increase the likelihood of health problems such as LBW and preterm birth and child disability. Furthermore, parents may have pre-established preferences for family size and for the mode of fertility control which may also correlate with preferences for child quality and investments in fetal and child health. Ignoring these heterogeneities in preferences may seriously bias the estimates of child health problems or disability on subsequent fertility decisions. The empirical strategies employed in the two studies using the NHIS/NSFG do not account for these unobserved preferences as they rely on comparing mothers of disabled and non-disabled children.

Fertility responses to unexpected child health shocks may be heterogeneous and may vary with preferences and socioeconomic characteristics. With regard to family size, one could argue that some parents would see the additional burden of raising a disabled child as a reason to stop having children, while others may decide to have more children in order to increase the proportion of non-disabled children in the household. Neither of these studies examine such potential heterogeneities, though MacInnes (2008) does acknowledge this limitation.³ We evaluate the heterogeneity in effects of child disability and health on subsequent maternal fertility by several demographic and socioeconomic characteristics.

³ The qualitative portion of MacInnes's (2008) study demonstrates this contrast in reaction to the delivery of a disabled child. One mother indicates that the slightly increased probability of the subsequent child having a disability changed the fertility plans of the couple, with the couple choosing to have fewer children in response to the birth of a disabled child. A second mother requested tubal

A further limitation of the previous work with the NHIS/NSFG relates to the parity of the child born with a disability, which was not fully captured in either of these studies. By design, the quantitative empirical analysis used by MacInnes (2008) only evaluates the fertility behavior based on the disability status of the first child. However, constraining analysis to the impacts of first child disability does not fully capture the effects of disability as many parents may be interested in having more than one child but may reduce total children as a result, where at the margin having a first less healthy child might matter less in their subsequent fertility decisions compared to the impact of a less healthy second or third birth on subsequent fertility. The empirical approach in Park, Hogan and Goldscheider (2003) addresses the existence of a disabled older child on the tubal sterilization of mothers after a subsequent birth, but the analysis combines the effect of a single versus multiple disabled children and does not model the effect of number of disabled children. An additional limitation of the approach of Park, Hogan and Goldscheider (2003) is that they only examine tubal sterilization as the fertility behavior. While the authors recognize this limitation, they claim the relatively low rates of male sterilization ameliorate this concern, suggesting that the only definitive stopping behavior would be one of these two options. However, examining the outcome of mothers' sterilization alone is not likely as informative in comparison to examining other subsequent fertility-related behaviors and outcomes as women and their partners who have not undergone sterilization have other options for limiting or stopping completed fertility (e.g. other contraception).

None of the previous studies evaluate the effects of at-birth health conditions such as preterm birth and LBW and only estimate the effects of child disability measures. As indicated above, both preterm birth and LBW may affect fertility decisions on their own not only because they significantly increase the likelihood of developmental disabilities and health problems

ligation immediately after delivering a disabled child near-term while a third indicated that despite her initial preference for two children, the disability of their second child led to a choice to continue expanding their family.

throughout childhood and later in life but also because they significantly increase healthcare costs and hospital length of stay and also reflect a higher-than-average risk of these outcomes to the mother in subsequent pregnancies. Indeed, the odds of a subsequent preterm birth and LBW child increase by 5.6 and 3.8 times, respectively, for mothers who have had a previous child with these conditions (Boyd et al., 2009; Khoury et al., 1989). Identifying the effects of these conditions on subsequent fertility decisions is also relevant because of their increasing prevalence as described above.

Another purpose for investigating the effects of at-birth outcomes is to evaluate the extent to which differences in timing of parental realization of child disability which is not measured may affect the effects of child disability on subsequent fertility. The authors of both of the previous studies using child disability as the sole measure acknowledge that the timing of realization of child disability could be impacting their findings. Specifically, disability in children may not be immediately apparent at birth or for years after the birth when subsequent children would have already been conceived. While this may imply an understatement of the effect of the presence of a disabled child on subsequent fertility, this was not empirically examined. Moreover, later realization of child disability might still affect fertility decisions if original target family or planned and/or preferred fertility spacing were sufficiently high and long enough.⁴

Finally, none of the previous evaluates the effects of child disability and health problems on birth spacing (i.e. conditional on having a subsequent birth). Identifying this effect is important as it may vary from the overall effect on whether to have a subsequent child or not. Child disability and health problems may affect parental decision in two ways: First, parents may update their preferences for having another child or not; second, among parents who want

⁴ Consider, for example, a woman who intended to have three children with intentions of these births spanning 4-5 years. Disability of the first child may not be realized by the time child 2 is conceived, but could be realized before 3 was conceived, leading to an adjustment of family size expectations.

to have another child, parents may decide to either shorten or extend the duration to the next birth depending on the expected utility increase relative to the shadow cost due to having another child earlier. Longer duration between births is known to decrease the risks of LBW and preterm birth (Conde-Agudelo et al., 2006; Zhu et al., 1999). Therefore, evaluating the child health and disability effects on birth spacing is highly relevant to understand their effects on population health.

III. Model

Theoretical Model

Our conceptual model is based on the children quality-quantity theory developed mainly in the work of Becker and Lewis (1973), Becker and Tomes (1976), and Becker (1991). In these models, the utility of the parents increase in quantity and quality of children. While these models are well known, it is worth revisiting the basic implications of the model for our work. Quality, which is represented by health in our work,⁵ is enhanced by parental contribution and investments in child health and by exogenous endowments, such as biologic, genetic and environmental factors. Assuming similar endowments among children, parents will invest equally in all children. In this case, the shadow prices of quality and quantity will depend on the quantity and the level of parental contribution to quality of children, respectively. Generally the comparative statics of this model imply that an exogenous increase in quality (specifically in parental contribution to quality) or quantity of children will increase the shadow prices of and

⁵ The model generally takes into account average child endowments, but it can also serve as a structure for analyzing the special case of impact of negative child health. Indeed Maclnnes (2008) appeals to this model (though not in a formal way) to examine the impact of disability of the first child on subsequent family decisions, where having a less healthy first child would be expected to decrease the propensity to further have children. However, focusing on the first child is limiting as the marginal utility of having non-disabled children may be greater than the shadow price of investment due to the presence of the unhealthy child. Conversely when the disabled child is born after a healthy child, the marginal utility of an additional healthy child (i.e. third or higher in parity) may be less than the shadow cost of the additional child due to the presence of the unhealthy child in the household.

decrease parental demand for quantity and quality, respectively. In an across-family comparison, and assuming the same level of child health endowment within a specific family, this implies that families with less endowed children will have fewer children compared to families with more endowed children due to the increase in the shadow price of number of children; achieving the desired level of quality requires an increased parental contribution to child health due to lower exogenous health endowment.

Formally consider the utility function $U(k, w, y)$, where y is a composite consumption good, w is a vector of the quality (health) levels of children, and k is the number of children. The price (p_q) of parental contribution to average quality per child depends on the marginal productivity of market and household quality-producing inputs, which is assumed to *increase* with increasing health endowments of the children in the family (e). In other words, p_q will increase with higher endowments due to the enhanced marginal productivity of parental investments. In contrast, a decrease in endowment would be expected to increase the price of an additional unit of parental contribution to child quality. The quality of children (w) in the family is assumed to be a function of parental contribution to quality (q), and child health endowment in the family (e): ($w = q + e$).

Parents face the following budget constraint: ($I = kqp_q + y$), where I represents family income.

The first order conditions of maximizing utility subject to the budget constraint are the following:

$$MU_k = \lambda qp_q = \lambda \Pi_k \quad (1),$$

and

$$MU_w = \lambda kp_q = \lambda \Pi_q \quad (2),$$

where $\Pi_k = (qp_q)$ and $\Pi_q = (kp_q)$ are the shadow prices of quantity and quality of children, respectively. Π_k depends on the amount of parental contribution to quality (health) and on the

price of that contribution. Π_q depends on the number of children in the family and on the price of contribution to quality.

With an exogenous increase in child health endowment, parents will invest less in w in order to increase their consumption as the same level of w can be obtained with lower parental contribution q . The marginal cost of contribution to quality would also decrease. Overall, the shadow cost of number of children will fall, increasing demand for the quantity of children.

This framework may be extended to study the effects of changes in health endowments on subsequent fertility decisions if parents update their expectation of the endowment of a subsequent k^{th} child based on the endowment of previously born children in the family:

$E(e_k) = f(e_{k-1}, e_{k-2}, \dots, e_1)$. If the endowment of the first child (e_1) as observed by the parents based on the child's health or disability status is higher than the initially "expected" endowment before the child's birth when parents are first considering an optimal number of children, then parents need to contribute less to quality, and the price of contributing to quality will decrease.

Anticipating similar endowments for future children, the shadow cost of additional children will fall, increasing demand for the quantity of children. However, if the endowment of the first child (e_1) is lower than "expected" endowment, then parents need to contribute more to quality of the individual child and the price of contribution increases due to the reduced productivity of parental contributions. This will reduce the expected endowment level of the second child $E(e_2)$, which will increase the expected marginal cost of having an additional child and may induce parents to stop fertility.

Consequently, if parents have a second child, they will re-evaluate their expectation of the endowment level of the third child based on the endowment levels for the first two children:

$E(e_3) = f(e_2, e_1)$. If the health endowment of the second child (e_2) is lower than that of the first

child (e_1), this will also reduce the expected endowment level for the third child, which in turn may increase the expected marginal cost of having a third child and so forth.

An exogenous decrease in child health raises the shadow cost of parental contribution to child health in several ways. Health problems and disability increase healthcare costs and the need for parental time inputs to care for the child. Disabled children use more hospital and physician services by about 8 and 5 times, respectively, than non-disabled children (Newacheck et al, 2004), which increase both out-of-pocket expenses and time costs. Child disability increases parental time contributions to child care by about 4 hours per day (Leonard et al., 1992).

Empirical Strategy

To analyze mothers' subsequent fertility choice we employ a discrete-time hazard model of the form used in previous studies of fertility behavior when the periods of observation are discrete time periods (e.g., Grogger and Bronars, 2001). We choose this model instead of a continuous-time hazard model as it allows us to exploit within-mother variation, which is needed to account for unobserved fixed effects such as preferences and endowments that may bias estimates that compare mothers of disabled and non-disabled children. The effect of interest is that of the number of previously born disabled or unhealthy children in the household, defined in one set of estimations based on activity-limitations (disability) and in other models based on low birth weight or preterm. We estimate these models separately for the probability of live birth, conception, and permanent sterilization, to evaluate whether there are potential differences in the effects of child health and disability on alternative fertility behaviors and outcomes. Consider the following model for the probability of observing a

fertility outcome (e.g. live birth) at time $\tau+1$ from the previous birth given that no outcome was observed at time τ :

$$P(t_i = \tau + 1 | t_i > \tau; D_i, S_i, X_i) = g(D_i, X_i, S_i), \quad (3)$$

where $\tau = 1, 2, \dots$ is the elapsed duration of the spell since last birth, D is an indicator for the number of previously born disabled children (or alternatively previous children born at low birth weight or preterm) for mother i , S is a measure of the dependence of the probability on the elapsed duration of the spell (spells restart after each live birth or pregnancy outcome, depending on the model), X_i is a vector of time varying household characteristics of household i at the time t including maternal age and the proportion of females among previously born children⁶, and u represent “unobserved” mother and household effects that are relevant to fertility decisions and outcomes and child health/disability. A mother enters the model after having a live birth with measurable health/disability status (depending on the child health/disability measure used) and contributes a sequence of 0/1 for the fertility outcomes and behaviors that we study. In the model for permanent sterilization, we censor women after they undergo sterilization. In the main specification for live births and conception, we do not censor women after sterilization which is on the causal pathway between disability/health problems and these fertility outcomes. However, in a sensitivity analysis, we censor these women after sterilization and find virtually similar results as described below.

Duration dependence (S) is an issue in this analytic framework. The length of the spell since the previous live birth (or pregnancy) would be expected to have an effect on the probability of observing such an outcome in a given year, and thus duration needs to be

⁶ We do not include marital status in the main model as it is one of the pathways through which child health and disability may affect subsequent fertility outcomes/behaviors. Furthermore, marital status was only measured at the time of pregnancy occurrence in the NSFG. However, we do estimate an additional specification where we adjust for marital status (assuming there are no marital status changes until marital status is measured again at pregnancy occurrence) and find virtually no change in the child disability/health effect.

accounted for in our model. We take a variety of approaches to represent S including estimating models with no adjustment for duration (i.e. excluding S), linear and log duration counts, and finally and most flexibly, we estimate the models where S includes a full series of dummies for elapsed duration. This approach is drawn from previous work by Grogger and Bronars (2001) on the duration to marriage or additional births of single mothers receiving welfare.

Because decisions related to fertility are likely influenced by unobserved preferences and endowment measures that affected both child health and disability as well as fertility (u), ignoring these unobservables could bias the effect of child disability on subsequent fertility (Rosenzweig and Wolpin, 1988). Therefore, we estimate the models using mother-fixed effects where only variation in number of disabled/unhealthy children for the same mother is used to estimate the effects of child disability and health problems on subsequent fertility decisions. Note that this model requires at least two live births for each mother to estimate this effect. The data provides adequate within-mother variation to estimate these models. In order to evaluate the potential magnitude and direction of bias resulting from the unobservable characteristics, we also estimate the models using mother-random effects. We estimate these models using a linear probability model (OLS) in order to directly retrieve the incremental effects on subsequent probability of outcomes for the fixed-effect models. All variance-covariance matrices are clustered at the mother level. We also estimate the models using logit (with both fixed and random effects) and find a similar pattern of results.⁷

We consider children to be disabled if they are unable to perform or are limited in kind/amount of major activity for their age or if they are limited in other activities. Major activity is defined as play and development for children who are less than 5 years and as school attendance and needs for children who are between 5 and 17 years. Children without any

⁷ The logit model results are available from the authors upon request.

reported activity limitation are considered as non-disabled. This definition is consistent with previous studies (MacInnes, 2008; Park et al., 2003).

The first model we estimate examines subsequent live birth as a function of previous children with disability. We demonstrate the difference in the estimated impact of mother random versus mother fixed effects models using various approaches to include elapsed duration, including linear count of the years elapsed since last birth, natural log of the years since last birth and finally a model which includes elapsed duration indicators. While the difference in the effects of the disabled children is small in comparison to the difference in effect size in the random versus fixed effect model, the latter model including dummy indicators for elapsed duration is preferred because it is most flexible way to account for duration effects in this framework, so this is the approach we use for the remaining estimations.

As discussed above, since some disabilities do not manifest until later in childhood, disability in previous children may not be realized until after subsequent fertility outcomes occur. Thus we consider a subsequent set of models which looks at LBW and preterm birth as signals of child health a mother or couple receives at the time of birth and whether these impact subsequent fertility outcomes. Children born at low birth weight and preterm birth are likely to develop lasting health and function limitations including schooling problems, neurodevelopmental delays, and long-term health problems such as cardiovascular diseases as well as reductions in human capital in the form of income or education (Anderson and Doyle, 2003; Currie, 2009; Frankel et al., 1996; Gluckman et al., 2008; Victora et al., 2008). Further, these outcomes are important on their own beyond their influence on child disability and development later in life given that they result in added health care costs and extended hospital length of stay. In addition, LBW and preterm children are likely to require large amounts of investment in the earliest periods of life, making the shadow cost of investment in quality apparent to parents immediately.

As discussed above, socioeconomic and demographic characteristics may modify the effects of child health and disability on fertility outcomes. Most of these characteristics are fixed in our models as constructed. To analyze what impacts these separate factors may be having on the relationship between existing children with disabilities and health problems on subsequent fertility outcomes we estimate the models with subsamples stratified by various mother characteristics at first birth. These include education (high school completion or less versus some college or more), income (at or less than 233% of federal poverty level (FPL) versus above), age at first intercourse (17 years old or younger versus 18 and older) and age at first birth (22 years old or younger versus 23 and older). Because those with less income face a higher shadow cost relative to more income in raising a disabled child, we expect that the effect size of disability on subsequent fertility should be higher. Education at first birth and age at first intercourse and first birth may also indicate available resources to allocate to child investment. On the other hand, less education at first birth, sexual activity at younger ages, and births at earlier ages are all potentially related to one another, and may be indicative of preferences for the present versus future, risk taking and less intentionality to determining subsequent fertility. Thus, whether there should be differences in relative effects size, their relative magnitude and direction is not immediately clear.

Rosenzweig and Wolpin (1988) found that child health endowment at birth had a impact on the likelihood of a subsequent live birth. However, the context of their study may not translate to behaviors of women in the US, due to differences in labor force participation intentions, differing social norms, etc. Because the qualitative work done by Maclnnes (2008) suggested a heterogeneous interfamily response to the birth of a disabled child, we also estimate a model that examines the time to next birth as a function of the outcome of the previous births in a mother fixed effects.

IV. Data

The research questions added in this paper require data on health of children born in the family as well as dynamic fertility decisions and outcomes. To our knowledge, no single US public use data file has those features, with both child health and complete fertility data are available. Thus, we employ merged data from the 1993 National Health Interview Survey (NHIS) and 1995 National Survey of Family Growth (NSFG) used in two previous studies (MacInnes, 2008; Park et al., 2003). The fertility and birth outcome data are from the NSFG, while the disability data are from the NHIS.

The NHIS is an annually conducted survey of a nationally representative sample of all households in the US (NCHS, 1995). Data is collected on household demographics and health status of household members. In 1993, data was collected on 43,007 households and 109,671 persons. Data is collected on presence of limitation in major activities that a person would perform as reported by the household interviewee. The major activity relates to the person's main activity in the last 12 months (NCHS, 1995). For children below the age of 5 years, major activity relates to development and play. For children between the age of 5 and 17 years, major activity relates to school attendance and needs. The specific data on schooling include whether the child is unable to attend school, attends (or needs to attend) special school/classes, or is limited in school attendance. Similar health data is available for the mother.

The NSFG provides data on complete fertility history of the mother including number and dates of pregnancies (including current pregnancy) and pregnancy resolutions (NCHS, 2000). Data is also collected on the mother sexual behavior, use of birth control techniques, and many other personal variables including health insurance, income, market work effort, history of marriage, and other demographic data.

The fifth cycle of NSFG conducted in 1995 used a sampling frame that was based on women in households that were interviewed in the 1993 NHIS. This enables the merging of the mother and her household records in the 1993 NHIS, including available health data on the children, to the 1995 NSFG record of the mother containing the complete fertility outcome and behavior data.

The study sample ranges from 3,775 to 5,539 unique mothers for the live birth (or conception) models that include disability and preterm birth, respectively. The panels for these mothers consist of 39,807 and 79,965 annual observations with complete data on all model variables. Only mothers who have had at least one live birth with measurable disability, LBW or preterm birth status are included. For each disability or health measure, only mothers who have had that measure reported for all their live births are included in the sample.

V. Results

Table 1 displays the summary statistics at the mother-year observation level and Table 2 shows a description of the live birth sample. About 6.5% of children are disabled, and about 7% and 8.8% were born LBW and preterm, respectively. Average maternal age at the child's birth is 25.2 years.

V.1 Effects on subsequent live birth and conception

The results of our discrete hazard model of the effects of number of previous live-born children with disabilities on the probability of a subsequent live birth estimated under different specifications for the duration of the spell and fixed and random mother effects are in table 3.⁸ Child disability has significant negative effects on subsequent live birth probability in all models. However, it is immediately apparent that the random effect estimates are substantially biased upward (toward zero) when compared to the fixed effects estimates (column 2). This suggests

⁸ Table A1 in the Appendix includes the full regression results for these models.

substantial bias in estimations that fail to account for unobserved preferences or health of the mother.

The overall pattern of results is generally insensitive to the different specifications for the duration of the spell, although the fixed-effect estimates are notably larger when spell-duration is not included. In that specification, the fixed effect estimation suggests that disabled children reduce the probability of a subsequent child being born by 0.21 per disabled child. Inclusion of spell length duration reduces the fixed-effect estimates; however, these remain large. In the most flexible framework including separate indicators for each period of elapsed spell year, disabled children reduce the probability of a subsequent live birth by 0.13 per disabled child based on the fixed-effects model. Comparatively, the random effect model suggests a decrease in subsequent live birth probability of 0.012 per disabled child. In what follows, we focus on presenting the effects of child disability and health problems using the most flexible specification that includes indicators for spell duration.

As mentioned above, the measures of disability in these data include conditions which wouldn't manifest until well after the birth of a subsequent child. This might understate the impact of the presence of a disabled child on subsequent fertility. Common at-birth health outcomes such as LBW and preterm birth may affect fertility behaviors and outcomes both through their effects on disability as well as directly due to the increase in healthcare treatments and time costs in at least the near term. As mentioned above, these birth outcomes are strong predictors of subsequent child disability. In our sample, preterm birth and LBW increase the probability of subsequent disability by about 3 and 4 percentage points respectively, which is equivalent to about half of the sample average rate of disability.⁹

⁹ These effects were comparable in random and fixed (mother-level) effects models. Detailed results are available from the authors.

As such, in subsequent estimations we also evaluate the impacts of previous LBW or preterm children on subsequent fertility. The results of these estimates are in table 4.¹⁰ Similar to the disability measure, these health problems have significant and comparable negative effects on subsequent live birth probability. Again, the random effect estimates are substantially lower and understate the effects of previous LBW or preterm children on subsequent live births compared to the fixed effect estimates, again suggesting there are unobserved preferences biasing previous estimates of the relationship between prior child health and subsequent fertility. Under the fixed effect model, previous LBW and preterm children reduce the probability of a subsequent live birth by 0.13 per affected child, which is virtually the same as the effect of disabled children. Under the random effect model, LBW and preterm children reduce this probability by 0.011. Given that early child disability is expected to result in larger declines in fertility compared to preterm birth and LBW because of the larger costs and needed investments, the similarity in effects between these conditions is theoretically consistent with the story that some disability does not impact subsequent fertility because it has not manifested prior to the subsequent birth, whereas preterm birth requires immediate investment to increase that child's health in the future.

Using live births subsequent to previous children with disabilities or health problems may not provide full insight into maternal fertility behavior due to planned or spontaneous abortions. Specifically, it is expected that the effects of child disability on health problems are larger on subsequent live birth than on pregnancy occurrence due to termination of unwanted pregnancies or spontaneous abortions that may partially reflect maternal reproductive health problems that may also be correlated to poorer child health outcomes. In the case of spontaneous abortions, using live births as the only fertility outcome may overstate the effect of previous child disability or health problems on the desire for subsequent children. If these

¹⁰ Table A2 in the Appendix lists the full regression results for these models.

reproductive health problems are time-invariant, they would not affect the mother fixed-effect estimates. However, these risks may vary over time. Furthermore, because both termination of pregnancy and miscarriages are expected to reduce the estimated impact of previous child disability and health problems on subsequent conceptions compared to live births, we are unable to disentangle the relative contribution of these factors to any differences we find between these fertility outcomes.¹¹ Nevertheless we estimate models for subsequent conception to see if either of these effects is present.

The resulting estimates of the model using subsequent conception as the outcome of interest are in table 5.¹² Similar negative and significant effects are seen compared to the effects on subsequent live-birth, with the random effect estimates being significantly lower (in absolute value) than the fixed effect estimates. The fixed effect estimates of previous disabled and LBW children on conception are slightly lower than those on live births (by about 1.2 and 0.6 percentage points, respectively), consistent with the theory. In contrast, the effect of preterm birth on conception is slightly larger than that on live-birth (by 0.4 percentage points). As a whole, these differences suggest minimal effects of selective termination of pregnancy or miscarriages on inference about fertility behaviors subsequent to child disability and health problems. The difference in effects for disability may suggest that time-varying maternal reproductive health problems may result in slightly overstating the effects of disabled or unhealthy children on subsequent fertility using live birth as the only fertility measure or that women may be terminating pregnancies in response to the presence of disabled or unhealthy children in the household.

¹¹ These data do include questions about spontaneous abortion and planned abortion, but the sensitivity of the latter and the fact that these data are self reported raises concerns about data reliability.

¹² Table A3 in the Appendix reports the full regression results for this model.

All the above analyses for effects of disability and health problems on subsequent live birth and conception do not censor women who become permanently sterilized. When censoring these women, we find virtually identical fixed-effect estimates, although the random effects become smaller and overall insignificant. Table A4 in the Appendix reports these results. This result suggests that most of the effects of child disability and health problems on subsequent conception and live births are not occurring through permanent sterilization decisions. Of course, the comparisons in effects between the uncensored and censored models may reflect different distributions of unobserved characteristics (such as preferences for risk taking) that may also modify the effects. Nonetheless, the result is consistent with women reducing their fertility outcomes as a result of child disability/health problems primarily through other pathways besides permanent sterilization.

V.2 Heterogeneity in Effects on Live Birth Probability

In table 6, we re-estimate the effects of child disability, LBW and preterm birth stratifying by fixed factors of mothers that would theoretically impact the observed relationships. These factors may impact fertility behavior independently, but these impacts were difficult to separate out in our models by virtue of the fact that the stratifying characteristics are time invariant in our data. As expected lower income level at first birth predicts a bigger effect of disability on subsequent fertility. Higher education and later age at first birth reduce the effect of child disability on subsequent fertility, consistent with the mother likely being more efficient in investing in child health to compensate for the disability or health shock effects. Lower age at first intercourse is indicative of a smaller effect of child disability on subsequent fertility, which may reflect more risk-tolerance and larger discount of the future.

V.3 Effects on Sterilization

We also evaluate if previous children's disability and health problems affect maternal "surgical" sterilization decisions using our discrete time hazard model. The purpose here is to evaluate if explore this as an additional fertility behavior and to compare the random-effect estimates of disability that ignore mother-level unobserved heterogeneity similar to those from a previous study with this data (Park et al., 2003) to the mother fixed-effect estimates in order to ascertain the degree to which these previous estimates may have been biased. In addition, we are able to examine the extent to which immediate birth outcomes may affect permanent sterilization behavior.

Table 7 reports the effects from this model. Disability, LBW and preterm birth have significant positive effects on permanent sterilization that are larger in the fixed than the random-effect models. In the preterm-birth models, the fixed-effect estimates are almost twice the magnitude of the random-effect estimates, indicating an increase in sterilization probability by 0.069 with each previous preterm birth. Again this suggests that even when measuring a 'hard' outcome of fertility stopping behavior unobserved maternal or household preferences or maternal health are likely impacting the result, leading to an understatement of the effect of child disability and health problems on subsequent fertility. LBW and preterm birth have a slightly larger on sterilization than disability in the fixed-effect model, suggesting that immediate birth outcomes may play an important role in the decision to become permanently sterilized beyond their effects on child disability.¹³

V4. Effects on birth spacing

Another aspect of fertility decisions that may be affected by the disability status and health of previous children is birth spacing, conditional on wanting more children. As

¹³ We are unable to estimate maternal fertility health changes resulting from the trailing birth with these data.

mentioned above, shortening the duration between pregnancies may have detrimental effects on child health by increasing the risks of adverse birth outcomes. Therefore, we evaluate the effects of disabled, LBW and preterm birth children previously born in the household on birth spacing at each subsequent live birth using both the random and fixed-effect estimations.

Table 8 reports these results.

The fixed-effect estimates suggest a decrease in time to next birth among women who had a subsequent live-birth after having children with disability, LBW or preterm birth by about 1 year. This is a large effect representing about 27-44% decrease in average birth spacing in the sample. The random effects are much smaller and insignificant for child disability and LBW. These results suggest that women who decide to have more children after having disabled, LBW or preterm birth children are likely to shorten the waiting time for having the next child. This may be due to a substitution effect if parents perceive a very high shadow cost of investment in children with disabilities or health problems that limits their ability to compensate for their health deficits, which may increase the desire to have another child sooner.

V5. Robustness checks

The fixed-effect estimates in our models are based on variation in fertility outcomes/behaviors for mothers who have had at least two children and have variation in the number of previous disabled, LBW or preterm birth children. Mothers whose first-born child is disabled (or alternatively LBW or preterm birth in the models using these health measures) but have no additional disabled children provide no variation into estimating the effects of child disability on subsequent fertility behavior using the fixed-effect models.¹⁴ Therefore, the fixed-effect estimates essentially apply to mothers who have at least two children and whose

¹⁴ These mothers provide within-mother variation for estimating the effects of control variables (maternal age and female proportion), which may affect the estimate of child disability. However, mother fixed-effect models that only include the previous number of disabled children without control variables provide the same estimates of disability effects if these women are excluded.

disabled (or LBW/preterm) child if they have only one affected child was not their first-born child. However, the estimated effects for these women may not apply to the overall population including mothers who have only one affected child who is also the first-born child. First-born disabled children in families with only one disabled child represent about 47% of all the disabled children in the study sample. Furthermore, the random-effect estimates presented above are in part based on between-mother variation in child disability from the subsample where the first child is the only disabled, LBW or preterm birth child. Therefore, the fixed- and random-effect estimates presented above may vary not only due to the role of time-invariant unobservable characteristics but also because they apply to different populations of women.

In order to evaluate the extent to which the sources of variation in child disability, LBW and preterm birth contribute to differences between the fixed- and random-effect estimates and the generalizability of the fixed-effect estimates, we re-estimate the above models excluding mothers for whom the first child is the only disabled, LBW or preterm birth child. If the random-effect estimates in this subsample are comparable to those in the full sample, then this would indicate that fixed unobservable characteristics are the main source of differences in results between the random- and fixed-effect models presented above and that the fixed-effect estimates may apply to women whose first child is the only disabled child.

Table 9 reports the effects of previous number of disabled, LBW and preterm children on subsequent live birth, pregnancy and sterilization outcomes excluding this subgroup. We also present fixed-effect estimates which virtually remain unchanged (some change very minimally due to changes in the effects of the control variables). The random-effect estimates for the effects of child disability, LBW and preterm birth on subsequent live birth and conception are lower (in absolute value) than those for the whole sample but remain statistically significant. In contrast, the random-effect estimates for permanent sterilization are larger than those using the whole sample, but are still lower than the fixed-effect estimates. As a whole, these results

suggest that the fixed-effect estimates are unlikely to be driven by excluding mothers whose first child is the only disabled child.

VI. Conclusion

We find significant effects of having previous children with disabilities or adverse birth outcomes including LBW and preterm birth on reducing subsequent maternal fertility. Models ignoring unobserved “fixed” heterogeneity such as preferences for health, risk taking, and health risks may significantly underestimate the disability effects on subsequent fertility. The results suggest a positive bias in random-effect estimates with women who are more fertile possibly having unobserved characteristics that increase their probability of having children with disabilities. Even though child disability and adverse birth outcomes increase permanent sterilization, most of the decline in subsequent conception and live births is due to other fertility or contraception behaviors. Interestingly, adverse birth outcomes including preterm birth and LBW have large and comparable effects on fertility behaviors compared to disability, suggesting that these outcomes are relevant for maternal and household decision making. The results highlight the importance of recognizing and addressing the burden of child health problems and disabilities on household behaviors including fertility and the implications for population health and growth and are informative for policy interventions.

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Table 1. Summary Statistics of Study Variables

Variable	Description	% of Mean (SD)	Number of observations [mothers]
Live birth*	0/1 indicator for a live birth during year t	9.1	39,807 [3,775]
Conception*	0/1 indicator for a pregnancy during year t	11.5	39,807 [3,775]
Sterilization	0/1 indicator for surgical sterilization during year t	3.6	32,669 [3,775]
Disabled*	Number of previous children with disabilities	0.13 (0.388)	39,807 [3,775]
LBW	Number of previous LBW children	0.15 (0.42)	62,916 [5,539]
Preterm	Number of previous preterm children	0.16 (0.441)	79,965 [6,792]
Maternal age*	Maternal age in years	30.0 (5.8)	39,807 [3,775]
Females*	Proportion of females among previous live births	0.47 (0.42)	39,807 [3,775]

Note: The Table reports the distribution of the study variables in the total sample. Descriptive statistics for selected variables (*) are reported for the panel consisting of 3775 mothers providing 39807 observations based on the subsequent live birth (or conception) model with child disability. The statistics for sterilization are based on the model for sterilization with child disability. The statistics for LBW and preterm birth are based on the live birth model that includes these health measures.

Table 2. Characteristics of the Live Birth Sample

Variable	% of Mean (SD)	N
% disabled	6.5	7401
% LBW	7.0	6676
% preterm	8.8	7398
Maternal age	25.2 (5.0)	7401
% females	48.1	7401

The descriptive statistics are based on total live births (including first birth) for mothers who have complete disability data for all their live births.

Table 3. Effects of Number of Previous Children with Disabilities on Subsequent Live birth Probability

No spell duration RE	-0.014*** (0.003)
No spell duration FE	-0.211*** (0.015)
Linear spell duration RE	-0.013*** (0.003)
Linear spell duration FE	-0.130*** (0.019)
Log spell duration RE	-0.014*** (0.003)
Log spell duration FE	-0.156*** (0.02)
Non-Linear (indicators for) spell duration RE	-0.012*** (0.003)
Non-Linear (indicators for) spell duration FE	-0.135*** (0.02)

Note: The table reports the effects of previous disabled children on probability of subsequent live birth under various specifications for the length of spell since last live birth and mother fixed and random effect estimations. The models adjust for maternal age and proportion of females, in addition to spell length variables as noted (Table A1 lists the full regression results). Clustered (by mother) standard errors are in parentheses; *** $p < 0.01$; RE=Random effects model; FE=Fixed effects model. The model includes 39,807 observations.

Table 4. Effects of LBW and Preterm Children on Subsequent Live Birth

	Random Effects	Fixed Effects
LBW	-0.011*** (0.002)	-0.128*** (0.016)
Preterm	-0.011*** (0.002)	-0.125*** (0.012)
<i>N</i>	62916	79965

Note: The table reports the effects from the discrete time hazard models with elapsed spell duration indicators and including maternal age and proportion of females. Clustered (by mother) standard errors are in parentheses; *** $p < 0.01$.

Table 5. Impacts of Previous Disabled, LBW or Preterm Birth Children on Subsequent Conception Probability

	Random Effects	Fixed Effects	N
Disability	-0.010*** (0.003)	-0.123*** (0.022)	39,807
LBW	-0.012*** (0.003)	-0.12*** (0.015)	62,916
Preterm	-0.011*** (0.003)	-0.129*** (0.012)	79,965

Note: The table reports the effects from the discrete time hazard models with elapsed spell duration indicators and including maternal age and proportion of females. Clustered (by mother) standard errors are in parentheses;*** $p < 0.01$.

Table 6. Impacts of Previous Disabled Children on Subsequent Live Birth Probability by Selected Characteristics

Group	RE	FE	Sample size
High school or less at first live birth	-0.013*** (0.004)	-0.15*** (0.028)	17670
Attended or completed college at first live birth	-0.011** (0.005)	-0.139*** (0.035)	13882
At or less than 233% of federal poverty line at first live birth	-0.01*** (0.004)	-0.155*** (0.028)	14936
Greater than 233% of federal poverty line at first live birth	-0.016*** (0.005)	-0.134*** (0.035)	16611
Age at first intercourse ≤ 17 years	-0.007* (0.004)	-0.13*** (0.023)	20685
Age at first intercourse ≥ 18 years	-0.016*** (0.005)	-0.15*** (0.039)	18638
Age at first live birth ≤ 22 years	-0.003 (0.004)	-0.161*** (0.026)	14706
Age at first live birth ≥ 23 years	-0.017*** (0.005)	-0.127*** (0.04)	16846

Note: The table reports the effects from the discrete time hazard models with elapsed spell duration indicators and including maternal age and proportion of females. Clustered (by mother) standard errors are in parentheses;*** $p < 0.01$.

Table 7. Impacts of Previous Disabled, LBW or Preterm Birth Children on Permanent Sterilization Probability

	Random Effects	Fixed Effects	N
Disability	0.047*** (0.008)	0.063*** (0.018)	32,669
LBW	0.041*** (0.006)	0.069*** (0.014)	49,738
Preterm	0.037*** (0.004)	0.068*** (0.01)	62,943

Note: The table reports the effects from the discrete time hazard models including maternal age and proportion of females. Clustered (by mother) standard errors are in parentheses;*** $p < 0.01$.

Table 8. Impacts of Previous Disabled, LBW or Preterm Birth Children on Birth Spacing in Years

	Random Effects	Fixed Effects	Average birth spacing in sample in years	N
Disability	-0.007 (0.09)	-0.9** (0.386)	2.04	3,626
LBW	-0.022 (0.1)	-0.97*** (0.35)	2.63	5,680
Preterm	-0.231*** (0.07)	-0.95*** (0.2)	3.51	62,943

Note: The table reports the effects of child disability and health problems on the time to the next live-birth for women who had a subsequent live birth. The models adjust for maternal age and proportion of females. Clustered (by mother) standard errors are in parentheses;*** $p < 0.01$.

Table 9. Impacts of Previous Disabled, LBW or Preterm Birth Children on Subsequent Fertility Outcomes Excluding Mothers Whose First Child is the only Affected Child

	Random Effects	Fixed Effects	N
<i>Effect on probability of subsequent live birth</i>			
Disability	-0.010*** (0.003)	-0.132*** (0.02)	37,119
LBW	-0.007** (0.003)	-0.128*** (0.016)	58,208
Preterm	-0.006** (0.003)	-0.125*** (0.012)	74,442
<i>Effect on probability of subsequent conception</i>			
Disability	-0.010*** (0.004)	-0.122*** (0.022)	37,119
LBW	-0.008** (0.003)	-0.12*** (0.015)	58,208
Preterm	-0.007** (0.003)	-0.129*** (0.012)	74,442
<i>Effect on probability of permanent sterilization</i>			
Disability	0.06*** (0.012)	0.064*** (0.017)	30,525
LBW	0.054*** (0.009)	0.069*** (0.014)	46,088
Preterm	0.053*** (0.006)	0.067*** (0.01)	58,458

Note: The table reports the effects from the discrete time hazard models with elapsed spell duration indicators for the live birth and conception outcomes and including maternal age and proportion of females. Clustered (by mother) standard errors are in parentheses;*** $p < 0.01$.

Appendix

TableA1. Linear Probability Regression Coefficients for Subsequent Live Birth Function with Previous Disabled Children

	No Duration RE	No Duration FE	Linear Duration RE	Linear Duration FE	Log Duration RE	Log Duration FE	Non-Linear Duration RE	Non-Linear Duration FE
Disabled	-0.014*** (0.003)	-0.211*** (0.015)	-0.013*** (0.003)	-0.130*** (0.019)	-0.014*** (0.003)	-0.156*** (0.020)	-0.012*** (0.003)	-0.135*** (0.020)
Maternal age	-0.009*** (0.000)	-0.013*** (0.000)	-0.008*** (0.000)	-0.037*** (0.001)	-0.009*** (0.000)	-0.034*** (0.001)	-0.008*** (0.000)	-0.037*** (0.001)
Females	-0.006 (0.004)	-0.035** (0.014)	-0.006* (0.003)	-0.016 (0.015)	-0.006 (0.004)	-0.021 (0.016)	-0.006* (0.003)	-0.016 (0.016)
Spell length			-0.003*** (0.000)	0.037*** (0.001)				
ln(spell length)					0.003* (0.002)	0.146*** (0.004)		
<i>Years since last live birth</i>								
2							0.100*** (0.005)	0.138*** (0.006)
3							0.093*** (0.006)	0.180*** (0.006)
4							0.075*** (0.006)	0.210*** (0.007)
5							0.051*** (0.006)	0.230*** (0.008)
6							0.038*** (0.006)	0.256*** (0.009)
7							0.028*** (0.006)	0.284*** (0.009)
8							0.021***	0.312***

							(0.006)	(0.010)
9							0.021***	0.347***
							(0.006)	(0.010)
10							0.025***	0.385***
							(0.006)	(0.011)
11							0.019***	0.414***
							(0.006)	(0.012)
12							0.020***	0.450***
							(0.005)	(0.013)
13							0.024***	0.488***
							(0.006)	(0.014)
14							0.024***	0.524***
							(0.006)	(0.015)
15							0.036***	0.571***
							(0.008)	(0.016)
16							0.023***	0.601***
							(0.005)	(0.017)
17							0.026***	0.642***
							(0.005)	(0.018)
18							0.027***	0.684***
							(0.005)	(0.019)
19							0.034***	0.728***
							(0.006)	(0.020)
20							0.031***	0.766***
							(0.006)	(0.021)
Constant	0.352***	0.530***	0.335***	1.061***	0.356***	0.953***	0.275***	1.037***
	(0.007)	(0.012)	(0.008)	(0.027)	(0.008)	(0.023)	(0.009)	(0.028)
<i>N</i>	39807	39807	39807	39807	39807	39807	39807	39807

Cluster robust (by mother) standard errors in parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

RE=Random effects model; FE=Fixed effects model. Omitted category for years since last life birth is one year.

Table A2. Linear Probability Regression Coefficients for Subsequent Live Birth Function with Previous LBW and Preterm Children

	LBW RE	LBW FE	Preterm RE	Preterm FE
LBW	-0.011*** (0.002)	-0.128*** (0.016)		
Preterm			-0.011*** (0.002)	-0.125*** (0.012)
Maternal age	-0.007*** (0.000)	-0.025*** (0.001)	-0.008*** (0.000)	-0.024*** (0.001)
Females	-0.006** (0.003)	-0.005 (0.013)	-0.006** (0.003)	-0.012 (0.011)
<i>Years since last live birth</i>				
2	0.096*** (0.005)	0.124*** (0.005)	0.103*** (0.004)	0.131*** (0.004)
3	0.092*** (0.005)	0.158*** (0.005)	0.094*** (0.004)	0.160*** (0.005)
4	0.066*** (0.005)	0.167*** (0.006)	0.067*** (0.005)	0.168*** (0.005)
5	0.050*** (0.005)	0.181*** (0.007)	0.054*** (0.005)	0.184*** (0.006)
6	0.033*** (0.005)	0.191*** (0.007)	0.031*** (0.005)	0.189*** (0.006)
7	0.027*** (0.005)	0.211*** (0.007)	0.029*** (0.005)	0.211*** (0.006)
8	0.020*** (0.005)	0.229*** (0.008)	0.021*** (0.005)	0.226*** (0.007)
9	0.015*** (0.005)	0.247*** (0.008)	0.016*** (0.005)	0.243*** (0.007)
10	0.015*** (0.005)	0.269*** (0.009)	0.017*** (0.005)	0.266*** (0.007)
11	0.007 (0.005)	0.284*** (0.009)	0.007 (0.004)	0.277*** (0.008)
12	0.013** (0.005)	0.311*** (0.010)	0.013*** (0.005)	0.303*** (0.008)
13	0.019*** (0.005)	0.338*** (0.010)	0.016*** (0.005)	0.327*** (0.009)
14	0.019*** (0.005)	0.361*** (0.011)	0.021*** (0.005)	0.353*** (0.009)
15	0.021*** (0.005)	0.384*** (0.012)	0.021*** (0.005)	0.373*** (0.010)
16	0.022*** (0.005)	0.407*** (0.012)	0.022*** (0.005)	0.396*** (0.010)
17	0.027*** (0.006)	0.434*** (0.013)	0.026*** (0.005)	0.421*** (0.011)
18	0.027*** (0.006)	0.458*** (0.014)	0.027*** (0.005)	0.442*** (0.011)
19	0.031*** (0.007)	0.485*** (0.014)	0.033*** (0.007)	0.470*** (0.012)
20	0.028*** (0.005)	0.504*** (0.015)	0.028*** (0.005)	0.487*** (0.013)

21	0.029*** (0.004)	0.527*** (0.015)	0.030*** (0.004)	0.510*** (0.013)
22	0.031*** (0.004)	0.554*** (0.016)	0.032*** (0.004)	0.535*** (0.014)
23	0.034*** (0.004)	0.579*** (0.017)	0.037*** (0.004)	0.559*** (0.014)
24	0.069*** (0.022)	0.637*** (0.027)	0.066*** (0.018)	0.611*** (0.023)
25	0.039*** (0.005)	0.632*** (0.018)	0.043*** (0.005)	0.608*** (0.016)
26	0.041*** (0.005)	0.659*** (0.019)	0.044*** (0.005)	0.633*** (0.017)
27	0.041*** (0.005)	0.683*** (0.020)	0.044*** (0.006)	0.655*** (0.019)
28	0.044*** (0.006)	0.709*** (0.021)	0.048*** (0.006)	0.677*** (0.021)
29	0.051*** (0.005)	0.740*** (0.021)	0.048*** (0.006)	0.717*** (0.017)
Constant	0.254*** (0.007)	0.673*** (0.019)	0.284*** (0.006)	0.666*** (0.016)
Observations	62916	62916	79965	79965

Note: The table reports the effects of LBW and preterm birth children on probability of subsequent live birth from the discrete time hazard models with elapsed spell duration indicators. Clustered (by mother) standard errors are in parentheses; * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$; RE=Random effects model; FE=Fixed effects model.

Table A3. Linear Probability Regression Coefficients for Subsequent Conception with Previous LBW and Preterm Children

	Disability - RE	Disability - FE	LBW - RE	LBW - FE	Preterm Birth - RE	Preterm Birth - FE
Disabled	-0.010*** (0.003)	-0.123*** (0.022)				
LBW			-0.012*** (0.003)	-0.120*** (0.015)		
Preterm					-0.011*** (0.003)	-0.129*** (0.012)
Maternal age	-0.009*** (0.000)	-0.040*** (0.001)	-0.008*** (0.000)	-0.026*** (0.001)	-0.009*** (0.000)	-0.025*** (0.001)
Females	-0.006 (0.004)	-0.022 (0.017)	-0.007** (0.003)	-0.007 (0.014)	-0.005* (0.003)	-0.006 (0.012)
<i>Years since last conception</i>						
2	0.052*** (0.007)	0.094*** (0.007)	0.046*** (0.005)	0.087*** (0.006)	0.049*** (0.005)	0.091*** (0.005)
3	0.032*** (0.007)	0.133*** (0.007)	0.035*** (0.006)	0.117*** (0.006)	0.031*** (0.005)	0.115*** (0.005)
4	0.002 (0.007)	0.155*** (0.008)	0.007 (0.006)	0.126*** (0.007)	0.006 (0.005)	0.126*** (0.006)
5	-0.021*** (0.007)	0.180*** (0.009)	-0.026*** (0.006)	0.125*** (0.007)	-0.025*** (0.005)	0.126*** (0.006)
6	-0.031*** (0.007)	0.212*** (0.010)	-0.033*** (0.006)	0.145*** (0.008)	-0.038*** (0.005)	0.141*** (0.007)
7	-0.046*** (0.007)	0.238*** (0.010)	-0.040*** (0.006)	0.164*** (0.008)	-0.044*** (0.005)	0.160*** (0.007)
8	-0.050*** (0.007)	0.272*** (0.011)	-0.044*** (0.006)	0.185*** (0.008)	-0.047*** (0.005)	0.180*** (0.007)

9	-0.053*** (0.007)	0.306*** (0.012)	-0.050*** (0.006)	0.204*** (0.009)	-0.057*** (0.005)	0.195*** (0.007)
10	-0.046*** (0.007)	0.349*** (0.013)	-0.050*** (0.006)	0.227*** (0.009)	-0.055*** (0.005)	0.218*** (0.008)
11	-0.047*** (0.007)	0.385*** (0.014)	-0.052*** (0.006)	0.248*** (0.010)	-0.056*** (0.005)	0.239*** (0.009)
12	-0.044*** (0.007)	0.425*** (0.015)	-0.046*** (0.006)	0.276*** (0.011)	-0.052*** (0.006)	0.265*** (0.009)
13	-0.042*** (0.007)	0.464*** (0.016)	-0.041*** (0.006)	0.304*** (0.011)	-0.049*** (0.006)	0.290*** (0.009)
14	-0.027*** (0.009)	0.516*** (0.017)	-0.038*** (0.006)	0.330*** (0.012)	-0.043*** (0.006)	0.317*** (0.010)
15	-0.033*** (0.008)	0.551*** (0.018)	-0.036*** (0.006)	0.354*** (0.012)	-0.043*** (0.006)	0.339*** (0.010)
16	-0.024** (0.010)	0.599*** (0.020)	-0.030*** (0.007)	0.383*** (0.013)	-0.038*** (0.006)	0.365*** (0.011)
17	-0.035*** (0.007)	0.632*** (0.020)	-0.026*** (0.007)	0.411*** (0.014)	-0.034*** (0.007)	0.392*** (0.012)
18	-0.035*** (0.007)	0.679*** (0.021)	-0.024*** (0.008)	0.436*** (0.014)	-0.030*** (0.007)	0.418*** (0.012)
19	-0.007 (0.027)	0.750*** (0.034)	-0.027*** (0.007)	0.459*** (0.016)	-0.034*** (0.006)	0.437*** (0.013)
20	0.031 (0.056)	0.827*** (0.061)	-0.020** (0.008)	0.491*** (0.016)	-0.028*** (0.007)	0.468*** (0.014)
21	-0.027*** (0.007)	0.814*** (0.025)	-0.027*** (0.005)	0.507*** (0.016)	-0.034*** (0.005)	0.486*** (0.014)
22			-0.023*** (0.006)	0.536*** (0.017)	-0.029*** (0.005)	0.514*** (0.014)
23			-0.009 (0.012)	0.575*** (0.020)	-0.017 (0.010)	0.550*** (0.017)

24			-0.000 (0.016)	0.608*** (0.023)	-0.009 (0.013)	0.580*** (0.019)
25			-0.011* (0.006)	0.621*** (0.019)	-0.016*** (0.006)	0.595*** (0.016)
26			-0.012* (0.006)	0.650*** (0.020)	-0.015** (0.006)	0.621*** (0.017)
27			-0.007 (0.007)	0.678*** (0.021)	-0.012* (0.006)	0.647*** (0.018)
28			-0.006 (0.009)	0.704*** (0.022)	-0.011 (0.009)	0.668*** (0.020)
29			-0.016** (0.007)	0.737*** (0.022)	-0.015** (0.008)	0.692*** (0.024)
30				0.000 (0.000)	-0.017*** (0.005)	0.732*** (0.019)
Constant	0.392*** (0.011)	1.183*** (0.032)	0.370*** (0.008)	0.778*** (0.020)	0.395*** (0.008)	0.764*** (0.017)
<i>N</i>	39807	39807	62916	62916	79965	79965

Standard errors in parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A4. Impacts of Previous Disabled, LBW or Preterm Birth Children on Subsequent Live Birth Probability Censoring Mothers after Permanent Sterilization

	Random Effects	Fixed Effects	N
Disability	-0.004 (0.004)	-0.139*** (0.022)	32,669
LBW	-0.007* (0.004)	-0.123*** (0.019)	49,438
Preterm	-0.006* (0.003)	-0.125*** (0.014)	62,943

Note: The table reports the effects from the discrete time hazard models with elapsed spell duration indicators and including maternal age and proportion of females. Clustered (by mother) standard errors are in parentheses;*** $p < 0.01$.