Reproductive Health and Labour Outcomes

A Thesis

by

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I, **Gauthier Tshiswaka Kashalala**, do hereby declare that this thesis is my own work and has not been previously submitted by me for the award of another degree at this or any other university.

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Reproductive Health and Labour Outcomes

Summary

This thesis formalizes the interaction between income and fertility outcomes using a fertility model taking account of mediation. Despite the diversity of studies concerned with the determinants of fertility, one consistent finding is that the timing and the number of children born to a couple are ultimately determined by the interaction between a couple's fecundity and their contraceptive behaviour. For example, fecund individuals may or may not be sexually active, may or may not be using contraception, which would influence their degree of risk for pregnancy. In this context, fecundity is a necessary but insufficient cause of fertility. The two variables, fecundity and contraception, are mediator variables in the causal path between income and fertility (Baron and Kenny, 1986). It follows that, understanding the interaction between biological and behavioural determinants of fertility holds the key to understanding fertility trends in any society.

With our understanding of causal-mediation in place, this thesis extends the health investment model to develop a model of the sequential nature of the human reproductive process. Because reproductive health is commonly understood as the capability to procreate and the freedom to decide if, when and how often to do so, poor reproductive health outcomes have long been acknowledged as a main cause of economic hardship for women and their children, accounting for 18% of the total global burden of disease and representing 32% of the burden among women of reproductive age. Thus, the presented theoretical model of the demand for reproductive health services

is a derived demand for reproductive health (with adjustment costs). Our theory suggests that the relationship between family planning services, their marginal costs, the stock of reproductive health capital and the woman's earnings volatility is essentially nonlinear. The theoretical predictions arising from the model are empirically supported from individual level South African data.

Because a number of the health costs are associated with 'mistakes', in the sense that childbearing might be ill-timed, because the reproduction process cannot be completely controlled, at least yet, the previous model is extended to account for uncertainty in the process. In other words, an assumption that contraception gives women perfect control over their fertility outcomes is considered to be too simplistic. Therefore, the focus remains on the effectiveness of contraceptive use in controlling fertility, but in a setting of uncertainty. The model developed fits nicely into the analysis of the sequential nature of the human reproductive process, as it makes use of the continuation or option value of contraception, which is assumed to depend on potential future labor market prospects, and the uncertainty inherent in the reproduction process. The model naturally leads to a mixed hitting time (MHT) empricial model; a woman becomes pregnant when the underlying stochastic reproductive health process first crosses a threshold in zero. We find empirical support for our theoretical analysis.

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The theoretical background to this thesis is hugely influenced by the discussions I had with James J. Heckman and his collaborators at the University of Chicago. I must also thank Jeff Racine for help with the incredible *np* package. Hakon Gjessing was prompt to fill me in on the practice of inverse Gaussian mixture model estimation. Thank you Alex Zimper. Your insightful comments are a testimony to your role as one of the pillars in the growing microeconomics wing of this department.

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Dedicated to my late mother Bernadette Mbuyi NKonga Mwa' Mbombo.

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Chapter 1

Introduction

1.1 A Puzzle Briefly Reviewed

This thesis revisits the inconsistency between the empirical negative fertility income effect and standard economic theory. The empirical evidence that fertility and standars of living are negatively related is inconsistent with standard consumer choice models of household fertility, which postulate that children are normal goods. If children were normal goods then the fertility income effect would be positive. Much study has addressed the determinants of the negative relationship between fertility and income. However, as yet, not a single explanation provided for this puzzle is universally accepted nor is close to being as robust as is commonly claimed (see Jones et al., 2011). For specificity, we illustrate our analysis using fertility and contraceptive calendar data from the 2000, 2004 and 2010 Malawi Demographic Health Surveys (MDHSs).

The impact of childbearing on a woman's earnings has two components. First, raising a child is a time-intensive endevour. In order to devote time to child care, a woman will have to forgo the opportunity to undertake income generating activ-

ities. If we assume that the opportunity cost of time is higher for individuals with higher earnings, higher earning women will tend to use more efficient contraceptive techniques in order to avoid their relatively high time cost of child care. As a result higher earning women will tend to have fewer children than their poor counterparts. Second, as noted by Mincer and Polachek (1974) and Mincer and Ofek (1982), work interruptions due to childbearing have a lasting negative effect on the woman's future earnings, through forgone experience and human capital depreciations. Furthermore, higher earning women also tend to be more educated and have access to a better information network, which put them at an advantage in more efficiently applying any specific contraception strategy they may choose. It follows that high income reduces family size through its positive impact on the choice of contraception efficiency level. Our work focuses on establishing this positive link.

The principal contribution of this study is to the derivation of a woman's optimal contraception strategy through a stochastic factor fertility model, which allows for the explicit modeling of the uncertainties surrounding both the family making process as well as the associated childbearing reward. Since family decisions are inherently sequential, (see Schmidt, 2008), we adopt the structural sequential stochastic framework pioneered by Heckman and Willis (1976). We construct a dynamic model of sequential fertility decisions for a utility maximizing woman, based on the expected utility framework of Beckerian models of fertility (Becker, 1960). At each decision node of this sequential framework, the woman is assumed to weigh the rewards of having a child now, against the benefit of postponing the arrival of the next child. In particular, given the uncertainty surrounding the human reproduction process, due to biological constraints on women's natural fecundity and other factors, there is an *option value* associated with the decision to postpone or stop childbearing.

Since having a child is an irreversible action, an increase in uncertainty will almost surely increase the continuation value associated with avoiding childbearing now (see Bernanke, 1983).

This study is motivated by the dynamic discrete choice models of Heckman and Vytlacil (2007). A key ingredient in our fertility model with associated reward outcomes is the underlying structure of a woman"s fertility decision-making process. We assume that the later is driven by the state variable represented by the woman's parity-specific option value of contracepting. As such, the model sheds light on how option values of postponing child birth determine contraception strategies at every birth interval. The main source of the option values here is the opportunity of accessing even higher earnings trough uninterrupted participation in the labour market. We show that the dynamics of the option value depend on the woman's natural ability to conceive as a latent stochastic factor, which takes into account the persistent variations in the physiological capacity to reproduce among women.

Our approach fits well with the sequential nature of the human reproduction process. In particular, we take advantage of stochastic control tools developed in the literature on stochastic optimization in incomplete markets (see Zariphopoulou, 2009) to capture the decisions to have children *as they are being made*, rather than focusing solely on realized fertility (see Iyer and Velu, 2006); we derive closed form solutions of the optimal contraceptive efficiency problem under imperfect contraception.

1.2 Analyzing this Puzzle: Some Preliminaries

The analysis of the interaction between fertility and measures of economic development is closely related to the literature on the demographic transition. There are suggestions from the demographic literature that fertility has been reduced by the

adoption of family planning services during demographic transitions (Coale, 1984). According to Coale (1984), three prerequisites should come to prevail for there to be a sustained reduction in fertility caused by the use of contraception: (i) fertility must be within the calculus of conscious choice leading to an optimal completed family size; (ii) effective techniques of fertility reduction must be accessible; and (iii) reduced fertility must be viewed as advantageous. However, based on the central assumption that the fundamental force behind fertility transition is the reduced parental demand for children brought about mainly by the changing opportunity costs of childbearing, current demand theories of the fertility transition only cover the first and third factors. This implies little to no attention being paid to the impact of contraception knowledge and availability.

Our approach unites all three of Coale's prerequisites for fertility decline by recasting the fertility-income debate into the income-contraception-fertility space. We argue that the puzzling negative effect of income on fertility is due to the lack of an explicit model of the demand for contraceptive efficiency within the current economic theories of fertility. Although a woman's probability of conception depends on external biological factors, actual fertility outcomes are stochastically explained by family planning choices and sexual behaviour. The biggest intellectual challenge then becomes the analysis of the determinants of the demand for family planning services and the subsequent impact of the latter on fertility.

The hypothesis is set forth that the interaction between a woman's fertility outcomes and her living standard is mediated by her contraceptive behaviour. If one considers contraceptive use to be the result of a conscious decision-making process, and a deliberate purposeful action on the part of a woman/couple, it implies that demand for family planning would provide a channel of choice, if not the only one,

through which economic factors such as income would be expected to explain observed fertility outcomes. In this setting, a rather interesting research theme revolves around the characterization of the way in which a woman/couple's contraception strategy evolves over its life cycle as a function of economic variables, such as the time paths of family resources, the parent's opportunity cost of time, the cost of contraception (see Heckman and Willis, 1976) and how this contraceptive behaviour, in turn, affects fertility outcomes.

We develop a fertility model that combines the literature on the human capital theory of the demand for health (see Grossman, 1972, 2000; Becker, 2007), with fertility transition theories (see Coale, 1984; Cleland and Wilson, 1987). The principal contribution of this study is to the consideration of family planning as an investment in reproductive health capital and its relation to women's labour market outcomes. In this framework, it is implied that contraception has an impact on both the standard of living and fertility outcomes. More specifically, there is a fundamental difference between health capital and other forms of human capital. While a person's years and quality of schooling and training may affect her productivity, good health can be viewed as a durable capital stock that determines the amount of time the person spends being productive (Schultz, 1961; Grossman, 1972; Strauss and Thomas, 1998). Given the fact that in the large majority of communities the burden of childbearing rests disproportionately on women, investment in family planning can also be considered to be an investment in a female-specific form of human capital. Conceptually, we assume that women with positive reproductive health outcomes are more productive, for longer, in the labour market.

So, in general, one should expect access to reproductive health services to be related to participation or performance in the labour market. Reproductive health

services encompass many areas, such as maternal health services and services related to sexually transmitted infections, but the analysis herein focuses on access to family planning services. In particular, delaying childbearing through birth control allows a woman to achieve higher levels of education and improves her time of successful activity in the labour market. Consequently, through contraceptive practice, a woman's fertility outcome is at least partially a result of conscious decision-making and a deliberate purposeful action, which provides researchers which a channel of choice to analyze the impact of economic variables on family-making decisions.

In what follows, we contend that for a woman participating in the labour force, the main objective of the investment in reproductive health is to avert any adverse effect on the accumulation of human capital that may arise due to mistimed, risky or unwanted childbearing. A pure reproductive health investment model would then suggest that a woman's optimal investment in family planning is driven by her desire to maximize her lifetime net money income production function, subject to the dynamics in the her own stock of reproductive health capital (see Ben-Porath, 1967). In this context, higher wages would induce investment in more efficient family planning services.

In this triangular framework, the role of family planning as an intermediate variable in the causal path between income and fertility can be investigated using causal mediation analysis (see Baron and Kenny, 1986; Pearl, 2001, 2012*b*; Imai, Keele and Yamamoto, 2010). Since competing scientific theories often imply that different causal paths underlie the same cause-effect relationship, the causal mediation analysis has the advantage of going beyond simple causal effects to provide the driving causal mechanisms (Imai, Keele and Yamamoto, 2010). We illustrate the estimation of certain aspects of our models using data from South Africa and the Democratic Republic

of Congo.

1.3 Analyzing this Puzzle: Some Empirical Commonalities

In general, the interaction between standards of living and fertility can be analyzed using one of the following three approaches: (i) the *Malthusian approach*, which concerns fluctuations in fertility in response to fluctuations in wages or productivity; (ii) the *time series approach*, which focuses on the time series properties of average fertility rates within a certain geographical area undergoing some sort of demographic transition; and (iii) the *cross-sectional approach*, which sets out to document heterogeneity within a cross-section of the population on the relationship between income and fertility (Jones and Tertilt, 2008).

Investigations interested in the Malthusian fertility cycles are among the oldest studies in the field of population economics. In his seminal work, Malthus (1993) predicted that fertility will remain at relatively high levels, such that rapid population growth would stifle improvements in the standard of living. However, this prediction failed to realize, and current versions of the Malthusian model have so far failed to produce a consensus around the direction of the relationship between fertility fluctuations and income. While some studies predict a positive correlation between income and fertility over the business cycle (Simon, 1969, 1977), others suggest that fertility may be counter-cyclical (see Butz and Ward, 1979).

Previous work using the time series approach has focused on the determinants of the *fertility transition*, as summarized in the evolution of the inverse relationship between a country's average fertility and average income in the same country. Starting

in the demography literature with Notestein (1945), the comparative roles of development and associated social changes, such as improved education and health, in explaining the marked decline in fertility have also been debated in economics. A common finding in both literatures is that decline in fertility coincides with the onset of sustained per capita income growth. The demography literature argues that the sustained per capita income growth produces a breakdown in the typical decision-making structure, which leads to a reversal of wealth flows within the family; originally that flow is from child to parent, but later from parent to child. In turn, this reversal, which is mainly due to individualistic desires of parents to promote the health and education of their children, makes children more costly to parents, leading to a decline in fertility (Caldwell, 1982). This approach is related to the economic fertility theory developed by Leibenstein (1975), which predicts a negative fertility-income link using the concept of status expenditures.

Nevertheless, the approach in the mainstream economics literature focuses on the significant role played by the opportunity cost of having a child. The most common explanation for the negative fertility-income relationship derived from these models is based on wage heterogeneity or differences in the tastes for children. Wages are considered to be the shadow price of time, and are highly correlated to income (Willis, 1973). The basic idea in this strand of literature is that, if childrearing is time intensive, the opportunity cost of time is very high for parents with higher wages. In other words, since the price of children is largely time-based, children will be more expensive for parents with higher wages, which should ultimately lead to fertility being negatively related to income (see Jones et al., 2011, for a review). In this framework, whether the completed family size is inversely or directly related to income then depends on the relative size of the income effect with respect to substitution effects.

The third empirical strand of literature, using cross-sectional data, has documented a largely negative relationship between income and fertility in developed societies, and a mostly positive link between the two in pre-industrial agrarian economies. For example, studies using data from South Korea and the U.S.A. find a negative relationship between fertility and income (or its surrogate economic status, occupational status and wealth) (see Westoff, 1954; Borg, 1989; Docquier, 2004). However, similar investigations find a positive relationship between fertility and proxies of economic status, such as wealth and farm size, using data from agricultural populations in England, France, and Prussia between the late 16th century and the end of the 19th century, as well as in modern Poland (see Wrigley, 1961; Haines, 1976; Simon, 1977; Weir, 1995; Clark and Hamilton, 2006).

One thing that has been rarely done in previous studies is to provide a comprehensive study of the fertility-income relationship using data from Sub-Saharan Africa. Fertility transitions in Sub-Saharan African countries, where agriculture is known to be a primary economic activity (see Diao et al., 2007), are not frequently analyzed. It is then tempting to conclude that by similarity to pre-transition Western agricultural economies, fertility would be positively related to measures of living standards. On the contrary, however, in recent years, demographic and health surveys have underlined declining fertility trends in many countries of sub-Saharan Africa.

To give a sense of the magnitudes, Figure 1.1 shows the declining trend in Malawi's Total Fertility Rate (TFR). The TFR is defined as the sum of the age-specific birth rates over all women of reproductive age, who are alive in a given year. Using birth history data collected in three successive DHSs from 2000 to 2010, we are able to compute period age-specific fertility rates and Total Fertility Rates following the Poisson regression procedure outlined in (Schoumaker, 2013).

Two noticeable features stand out from the figure. The first is the discrepancy in the fertility data during the period from 1995 to 2000, which points to potential weaknesses in the way birth histories are collected through the DHSs. In particular, there appears to be a downward bias in fertility rates resulting from possible displacements and/or omissions of births that happened at the beginning of the birth calendar in the 2000 and 2004 surveys. The birth calendar in a typical DHS covers roughly a period of 5 years before the interview. What the computations show is that there is an under-estimation of more than one child at the beginning of the birth calendar, compared to the estimation from the immediately following survey. The second feature that stands out is the substantial reduction in overall fertility over the last two decades in Malawi. From just above 8 children per woman in 1980, fertility fell to 5.5 twenty years later.

However, the average value of TFR summarizes the evolution of fertility in a way that completely abstracts from the diversity in experiences among women. By considering only the average rate of fertility in isolation, one completely misses some crucial information contained in the distribution of children across women from different socio-economic backgrounds. Understanding human fertility requires, among other things, biological and behavioural factors, which would benefit from the conceptualization of intermediates in the pathway to conception or birth (Louis, 2011). Before outlining our model in the following chapter, we provide a brief synopsis of some of the main features on the empirical literature. We begin with a composite measure of a household's cumulative living standard as expressed by the DHS wealth quintiles.

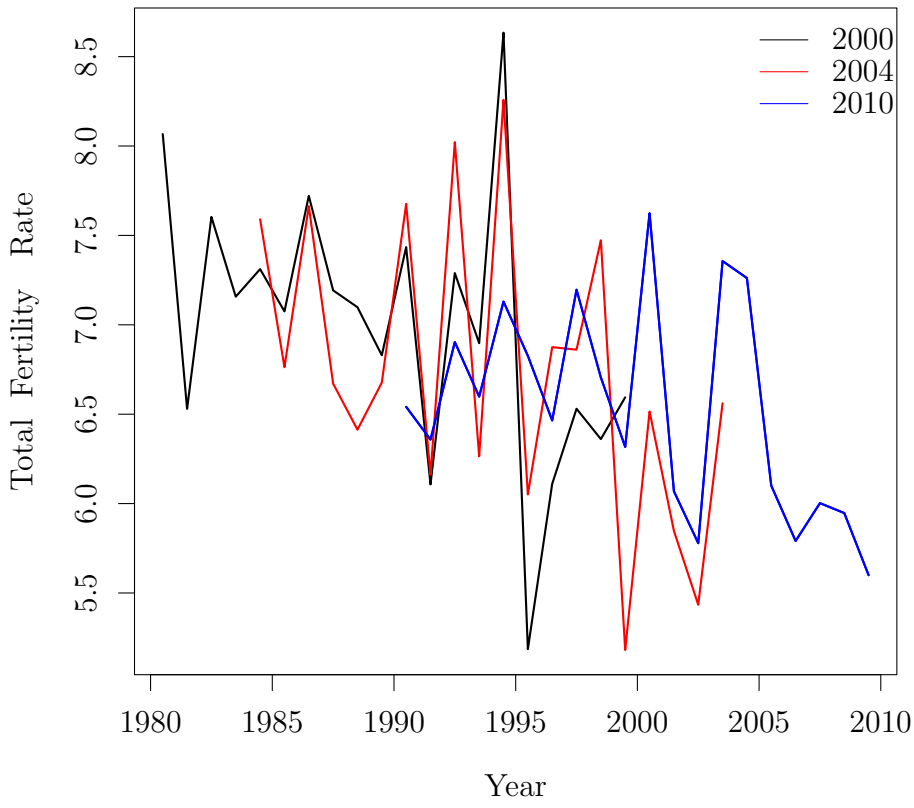

Figure 1.1: *Total Fertility Rate* (15 – 49) for the 20 calendar years preceding each survey, Malawi 2000, 2004 and 2010 DHS

1.3.1 Fertility and Wealth

The link between fertility and wealth may be positive or negative. In his wealth flow theory of fertility decline, Caldwell (1976) argues that the fundamental issue in demographic transition is the direction and magnitude of intergenerational wealth flows. When the flow of money, goods, and resources is from the younger to the older generation, an increase in wealth will result in higher fertility rates. But when the flow of wealth is from parents to children, being childless is the most rational economic

behaviour. However, for social and psychological reasons, couples would procreate though they have many fewer children. In this situation, wealth is negatively related to fertility.

Table 1.1 includes TFR for women living in *poorest*, *poorer*, *middle*, *richer* and *richest* households, respectively. Typically, the DHSs do not include traditional economic measures, such as consumption expenditure, but they collect information on ownership of durable assets that are thought to have direct influences on health, leading to the development of a wealth index (see Filmer and Pritchett, 2001; Howe et al., 2009). The asset-based DHS wealth index better represents long-term or permanent economic status and performs as well as the more traditional indexes of consumer expenditures (see Wagstaff and Watanabe, 2003).

The table shows that women in poor households have a higher number of children than in rich families, which suggest the presence of a negative link between fertility and income. The number of children per woman in poor households is nearly double that of a woman in richest households. Those women in the poorest and poor households seem to have the same level of fertility rate at 6.8 children per woman. The composition of the wealth index provides only a hint towards our understanding the negative correlation between fertility and the DHS wealth index.

The list of assets and services currently queried in the standard DHS questionnaires for the calculation of the wealth index includes a question about ownership of agricultural land and size (Rutstein, 2008). Other things being equal, differentials in fertility by household land ownership status contain information about optimal reproductive behaviour, based on differential demand for children and the services they provide to parents. In particular, land appears to have at least two distinct aspects that influence fertility in opposite directions. One is the size of land a household

controls for cultivation purposes, including leased land and excluding owned land rented out. A second dimension is land ownership, which refers to the system of land distribution and produce from the land (Cain, 1985).

It has been suggested that the size of the area cultivated by a family affects fertility by modifying the relative cost of an extra child. In this case, families with access to larger holdings are able to use additional family labour more profitably, which leads to higher fertility. It follows that, the complementarity of child labour and landholdings underlies the positive relationship between farm size and fertility. On the other hand, ownership and operation of land not only entails returns to labour and management accruing to cultivators of rented land, it also provides an equity return. Consequently, land ownership should reduce the importance of children as sources of parental security in old age and contribute to lower fertility. Thus, the negative relationship between land owned and fertility (Cain, 1985; Stokes et al., 1986).

It follows that the negative correlation between the wealth index and the TFR presented in Table 1.1 may be explained by the following factors: (1) importantly, wealth flows towards investments in children (*e.g.* education and health); and (2) higher income returns to equity captured by landowners and the consequent increase in old-age security.

1.3.2 Fertility and Settlement

Another, less discussed, aspect of fertility closely related to socio-economic status is differences across settlements, including urban-rural fertility variations. Studies show that significant differences in fertility levels between various settlements persist: the larger the settlement, the lower are the fertility levels (Kulu, 2013). Table 1.2 illustrates the Malawi's case, and suggests that fertility rates in the country are higher

	Total Fertility Rate			
Wealth Index	Coef.	Std. Err.	z	$P > \|z\|$
Poorest ($n = 4,524$)	6.820	0.138	49.48	0.000
Poorer ($n = 4,487$)	6.820	0.139	48.91	0.000
Middle ($n = 4,701$)	6.310	0.130	48.57	0.000
Richer ($n = 4,681$)	5.253	0.115	45.81	0.000
Richest ($n = 4,534$)	3.749	0.098	38.16	0.000

Sample size, $N =$ 22,929.

Source: 2010 Malawi DHS.

Table 1.1: Fertility and Wealth

in small towns and rural areas. Two competing hypotheses regarding spatial fertility variation have been researched in the literature.

First is the *compositional* hypothesis, which suggests that fertility levels vary between settlements for the simple reason that different people live in different places. The share of single people is larger in large cities. Since marital status is correlated to childbearing, over-representation of single people in large places may explain the lower fertility rates in large cities (Hank, 2002). In this context, selective migrations may also explain variations in spatial fertility, if we assume that couples who intend to raise a family may move from larger cities to smaller ones, because the latter are perceived as better suited to raising children (Kulu and Boyle, 2009). And in the context of sub-Saharan Africa, where the massive and growing labour migration phenomenon is reshaping the reproductive landscape, spousal separation decreases exposure to conception, leading to lower marital fertility in rural areas, at least in the short term (see Bongaarts et al., 1984; Kulu, 2013). Using data from Mozambique, a

recent analysis of the lifetime fertility of rural women married to migrants shows that it varies directly with the time spent in migration by their husbands (Agadjanian et al., 2011).

Second is the *contextual* hypothesis. In this literature, the factors related to the immediate living environment of individuals plays a crucial role in determining fertility levels. It is assumed that the context influences people's fertility behaviour mainly through economic and cultural factors. A rural town's population is often considered to have a *family-oriented* subculture, while large cities are largely assumed to promote individual autonomy and self-actualization, which usually requires fewer children. In this setting, couples willing to form a family will tend to stay in small towns, whereas those with no childbearing ambitions relocate to larger cities (see Lesthaeghe and Neels, 2002). Furthermore, compared to rural areas, large cities are known to be relatively expensive places to raise children. This is not only because of the high cost of food, commodities and services, but also due to higher opportunity costs of time in cities (Becker, 1991; Michelin, 2004). The urban-rural fertility variation may also be explained by cultural factors, which determine people's attitudes towards family formation.

	Total Fertility Rate			
Residence	Coef.	Std. Err.	z	$P > \|z\|$
Rural ($n = 19,872$)	6.079	0.062	98.58	0.000
Urban ($n = 3,055$)	4.040	0.126	32.14	0.000

Sample size, $N =$ 22,929.

Source: 2010 Malawi DHS.

Table 1.2: Fertility and Residence

1.3.3 Fertility and Education

Almost every discussion of fertility differentials makes reference to the role of education in reproductive decisions. Moreover, the compositional factors proposition, discussed in the previous section, that fertility levels vary between places because different people live in different settlements, may actually be an indication of differentials in the levels of education. For many countries, the share of highly educated people is larger in cities than in small towns and rural areas, such that lower fertility in rural areas is mainly the expression of low levels of education (Kulu, 2013). Table 1.3 suggests that educational composition is an important determinant of fertility variation in Malawi. It appears that fertility levels in the country are lowest for highly educated individuals and highest for individuals with no formal education.

A number of studies have used education as a proxy for lifetime income, making the literature on the interaction between education and fertility similar in many ways to that on income and fertility (Jones and Tertilt, 2008). From a theoretical standpoint, the role of education in fertility decline has been analyzed with reference to Coale's (1984) preconditions for fertility transition mentioned earlier. In particular, the influence of education is closely related to the first two prerequisites that concern the individual's ability to control the human reproductive process (Cleland and Wilson, 1987). The main idea is that the growth of formal education brought about by the structural modernization of societies has created a psychological shift that empowers individuals with a sense of control over their destiny and pursuit of achievement. Such a general shift in outlook would change individual's attitudes towards and increase the propensity to use birth control.

There is overwhelming evidence to support the idea that, during transition, high levels of parental education are associated with lower marital fertility through greater

use of family planning services. In particular, borrowing from the literature on the relationship between mother's education and infant mortality, more recent studies have found that, not only does the education of the mother exert a strong influence on infant mortality (Caldwell, 1979), it appears to be a very strong predictor of the decline in fertility, as well (see Schultz, 1986; Preston and Hartnett, 2010). This link tends to be relatively stronger compared to other purely economic covariates, such as income and occupational status. In most countries, a few years of schooling are sufficient for a shift in reproductive behaviour, through changes in perceptions, ideas and aspirations (Cleland and Wilson, 1987). Thus, the prediction that, as countries adopt universal primary schooling, fertility would eventually fall, naturally (Caldwell, 1982).

The literature also does differentiate between the fertility impact of the level of education received by parents themselves, from the influence of schooling opportunities of their offspring. The growth of educational opportunities for children encourages investment in the quality of children, as opposed to the quantity (Becker and Lewis, 1973). If children are indeed normal goods, then quantity should change directly with income. In Becker's *quantity-quality* model of fertility choice a parent has to decide between having a large number of children with few economic opportunities each, or have few children with a large bequest, or large investment in education. Thus, it is not surprising if, at the level of the individual family, aspirations for the education of children are often found to be negatively correlated with overall family size desires (see Cleland and Wilson, 1987; Jones and Tertilt, 2008).

	Total Fertility Rate			
Education	Coef.	Std. Err.	z	$P > \|z\|$
No Education ($n = 3,390$)	6.920	0.172	40.18	0.000
Primary ($n = 15,252$)	5.888	0.069	85.40	0.000
Secondary ($n = 3,964$)	3.771	0.117	32.17	0.000
Higher ($n = 321$)	2.094	0.272	7.71	0.000

Sample size, $N =$ 22,929.

Source: 2010 Malawi DHS.

Table 1.3: Fertility and Education

1.4 Contributions

The study attempts to recast the debate around the fertility-income puzzle into the income, birth control and completed family size space, with the decision of how much to spend on family planning services treated as an investment decision. The treatment of the cost of contraception as an investment decision in human capital, whose returns are subject to uncertainty, is the principal contribution of this work. The interest is in the factors that influence the time at which a woman chooses to contracept or not; and if she chooses to contracept, what would be her optimal level of contraception efficiency. Happel et al. (1984) argue that, in an environment where the expected average number of children is a societal norm, analyzing timing of births is as significant as investigating completed family sizes.

Similar types of research questions have been tackled before using real options reasoning borrowed from the literature on financial modeling (see Dixit and Pindyk, 1994). An option is the right but not the obligation to take an action in the future. Using the real options approach to analyze contraception as investment has the merit

of describing the contracepting process as it unfolds, and explicitly incorporate uncertainty to the process in a more natural way. In the case at hand, the theoretical framework argues that the influence of uncertainty on the contraception behaviour is reflected in the stochastic nature of the *option* to continue using birth control services.

Two main sources of uncertainty are considered: the woman's natural ability to fall pregnant, *i.e.* natural fecundity, and the opportunity cost of childrearing. Assume for simplicity that stopping contraception coincides with the woman getting pregnant, and that all pregnancies result in live births, then considering contraception as an investment helps to address the question of whether there is any value in postponing to have the next child, which may arise as a response to the influence of uncertainty (see Iyer and Velu, 2006). As noted above, this is a very important question in Africa where fertility transition is happening through the use of contraception to postpone childbearing rather than to space births or limit the family size (see Timaeus and Moultrie, 2008; Moultrie et al., 2012). Even in non-African settings this approach can still be of interest.

The argument above places changes in the continuation value of contraception at the centre of the proposed economic fertility model. The continuation value of contraception is defined as a latent stochastic process which describes the dynamics of the net expected utility of contracepting. The essence of the argument is that having a child is like the decision to exercise an option. A woman is assumed to weigh short-term costs of contraception against net benefits of the next child and choose the level of contraception efficiency that maximizes welfare. As a result, depending on the value of the latent stochastic process hitting a threshold, the woman can decide at any time to stop birth control and have a child or to postpone the decision and continue using contraception (see Stokey, 2009). It follows that the latent stochastic

process drives all theoretical and empirical results derived from the economic fertility model proposed here.

The primitives of the theoretical model yield implications for fertility behaviour which are subject to empirical investigation. Discussions in the empirical analysis focus on providing an empirical content to the theoretical prediction that income should be positively related to fertility. Assume that a woman stops contracepting when the continuation value first hits a threshold, and the distribution of associated time-to-event is well known in closed form, then data on birth intervals can be empirically analyzed using relevant mixed hitting time duration models Aalen et al. (2008); Abbring (2012).

For example, we postulate that contraception has an influence, not only on fertility outcomes, but also on the labour market performances. It follows that family planning services mediate the causal relationship between fertility and labour market outcomes. We trace out these causal paths using a causal mediation effect model (Pearl, 2001; Imai, Keele and Yamamoto, 2010; Imai, Keele and Tingley, 2010; Pearl, 2012*b*) using DHS data from Malawi. Table 1.4 indicates that the total fertility rate among women using modern contraceptive methods in Malawi is almost half that of women using traditional methods or not contracepting at all.

Furthermore, data from the reproductive calendar show that, in the five year immediately preceding the 2010 Malawi DHS, a steady increase in the average level of contraceptive efficiency was accompanied by a corresponding decline in the total fertility rates. As illustrated in Figure 1.2

The empirical framework also redefines the statistical threshold regression framework (see Aalen et al., 2008; Pennell et al., 2010) within the economic framework of theory of choice using nonseparable models in unobservables (see Florens et al.,

	Total Fertility Rate			
Residence	Coef.	Std. Err.	z	$P > \|z\|$
Rural $(n = 19,872)$	6.079	0.062	98.58	0.000
Urban $(n = 3,055)$	4.040	0.126	32.14	0.000

Sample size, $N =$ 22,929.

Source: 2010 Malawi DHS.

Table 1.4: Fertility and Residence

2008; Imbens and Newey, 2009). The resulting mixed hitting time duration model, with selection, is used to estimate the returns to contraception under essential heterogeneity. The model is consistent with the analysis in Abbring and van den Berg (2003), and is related to the dynamic discrete choice model outlined in Heckman and Navarro (2007) and the analysis in Abbring (2012). However, it complements the dynamic discrete choice model by exploring the case of a continuously distributed treatment in continuous time; and extends the mixed hitting time model described in the latter by allowing the agent some control over the dynamics of the entire system through the dependency of the drift of the latent stochastic process on the demand for contraception efficiency, the choice variable.

1.5 African Exceptionalism

Building on the work of Heckman and Willis (1976), the study derives an integrated theoretical and empirical framework aimed at analyzing the interaction between contraception and labour market outcomes. The main hypothesis is that the causal relationship between fertility and income is mediated by the demand for con-

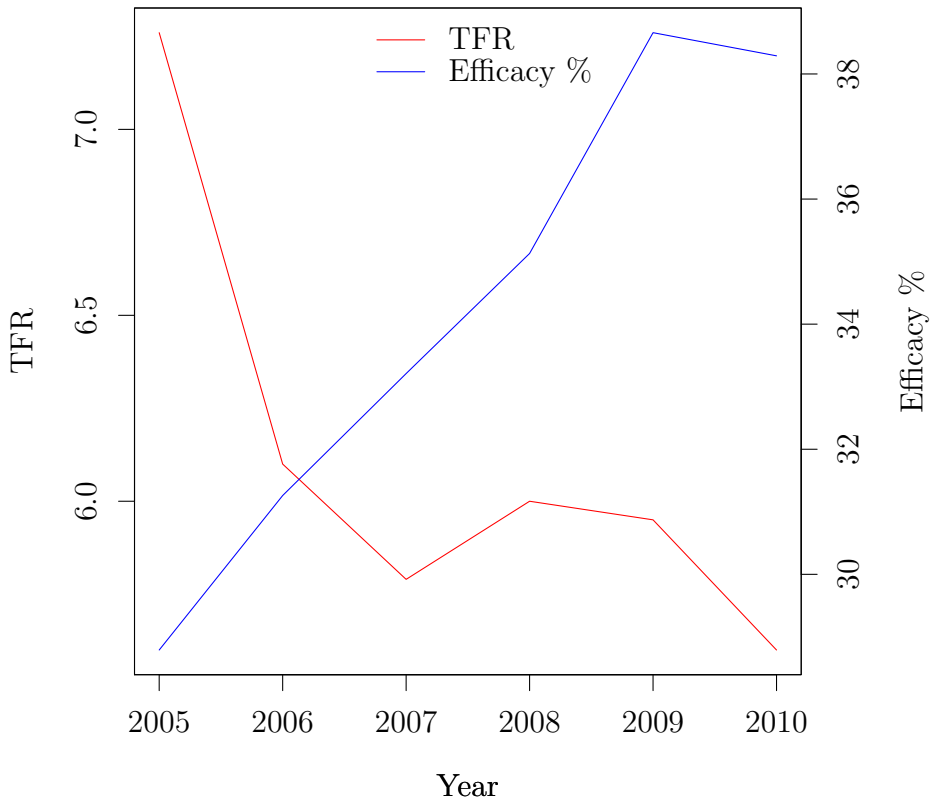

Figure 1.2: Total Fertility Rate (15 – 49) for the 20 calendar years preceding each survey, Malawi 2000, 2004 and 2010 DHS

traception efficiency. Assuming, as any standard Beckerian fertility framework, that children are a normal consumption good, the analysis focuses on fertility decision-making process at the margin, where a woman could rationally choose whether or not to stop contraception and have the next child. The sequential approach adopted in this study is consistent with the suggestion that birth postponement, rather than completed family size, is a decisive factor in the family formation decision-making process in many contexts in Africa (see Caldwell et al., 1992; Johnson-Hanks, 2007; Moultrie et al., 2012). This makes the choice of the African continent to investigate

the interactions between reproductive health outcomes and labour market outcomes a subject of particular interest in its own right.

Indeed, the choice is dictated by the pivotal role that family planning services play on the demographic transition unfolding across the continent. There is a growing body of evidence, emerging from the Demographic and Health Surveys (DHS) program, which suggests that falling national fertility levels since the 1990s are largely due to a rising proportion of demand for fertility control specifically directed towards increasing birth intervals (see Caldwell et al., 1992; Johnson-Hanks, 2007; Moultrie et al., 2012). This phenomenon extends the motivation for contraceptive practice beyond the standard limitation of family size, and has led to a particular form of fertility transition characterized by similar fertility declines across all parities and age groups in Africa (see Caldwell et al., 1992; Moultrie et al., 2012).

Moreover, South Africa presents a curious case of fertility transition accompanied by persistent high unemployment and poverty levels. In particular, one of most remarkable trends in family life in the past two decades has been the rise in both birth intervals and female unemployment rates. Despite the fact that fertility rates have been slowly declining over the past four decades, with the projected median birth intervals approximatively doubling from 33 months to more than 60 months (see Timaeus and Moultrie, 2008), female unemployment has risen from around 20% in 1995 to more than 30% in 2005 (Kingdon and Knight, 2007; Banerjee et al., 2008).

The data from South Africa is provided by the Cape Area Panel Study (CAPS) Integrated Waves 1-2-3-4, which collected information from adolescents in the metropolitan Cape Town, South Africa four times between 2002 and 2006[1] (Lam et al., 2008).

[1]The Cape Area Panel Study Waves 1-2-3 were collected between 2002 and 2005 by the University of Cape Town and the University of Michigan, with funding provided by the US National Institute for Child Health and Human Development and the Andrew W. Mellon Foundation. Wave 4 was collected in 2006 by the University of Cape Town, University of Michigan and Princeton University. Major

The data set is an unbalanced panel of 4752 randomly selected young people from 3312 households who were 14-22 year old in August-December, 2002.

Among the several interesting variables in this data set are the variables from the module which collected information on sexual and reproductive health aspects of adolescence. Of particular importance, for the purpose of this study, are the answers to the question of whether or not the young female did use contraception at last sexual intercourse, and the follow up question on what contraception method did she use. In addition, the sample is restricted to young adult women in the first and the last two waves because of the close similarity between the two respective questionnaires.

The failure of the DRC's fertility to decline until now is the main justification behind the inclusion of this country into the analysis. A major goal in doing so is an attempt to contribute to the understanding of the reasons why fertility transition has so far eluded the DR Congo. According to recent research, while most countries in Africa have completed or are well advanced in the transition to low fertility, the DR Congo is still far from meeting conditions for a sustained fertility transition (see Romaniuk, 2011). With a total fertility rate estimated at above 6 per woman (2007 Democratic Republic of Congo's Demographic and Health Survey), the DRC is a high fertility country. One of the reasons for the high fertility rates has been the low level of modern contraceptive methods use (see Kayembe et al., 2006). Also, in contrast to the situation in South Africa, these stubbornly high fertility rates in the DRC coexist with high unemployment.

The information on reproductive behaviour in the DR Congo comes from the DHS for the year 2007. In the data set contraception methods are classified as *traditional*, *folklore* and *modern*. Traditional methods include Periodic Abstinence (also known

funding for Wave 4 was provided by the National Institute on Aging through a grant to Princeton University, in addition to funding provided by NICHD through the University of Michigan.

as Rhythm), Withdrawal and Abstinence. Modern methods include Pill, IUD, Injections, Diaphragm, Condom, Sterilization, Implants, Foam/Jelly and Lactational amenorrhea. Folkloric methods include all the methods not specifically mentioned but believed to be less efficient than the traditional methods. If a woman has used both a traditional method and modern method then the latter takes priority and she is coded as she has used a modern method. In the same vein, a woman who is recorded to have used a traditional method if she has used both a traditional method and folkloric method.

1.6 Organization

In first analysis chapter of this thesis, we formalize the interaction between income and fertility outcomes using a fertility model taking account of mediation. Despite the diversity of studies concerned with the determinants of fertility, one consistent finding is that the timing and the number of children born to a couple are ultimately determined by the interaction between a couple's fecundity and their contraceptive behaviour. For example, fecund individuals may or may not be sexually active, may or may not be using contraception, which would influence their degree of risk for pregnancy. In this context, fecundity is a necessary but insufficient cause of fertility. The two variables, fecundity and contraception, are mediator variables in the causal path between income and fertility (Baron and Kenny, 1986). It follows that, understanding the interaction between biological and behavioural determinants of fertility holds the key to understanding fertility trends in any society.

In the second analysis chapter of this thesis, we undertake to model the contraception side of the argument presented in the chapter immediately preceding it; however, it is placed within a model of health investments. Because reproductive health is com-

monly understood as the capability to procreate and the freedom to decide if, when and how often to do so, poor reproductive health outcomes have long been acknowledged as one of the main causes of economic hardships for women and their children (see Schultz, 2008).[2] The analysis in this chapter describes a theoretical model of the demand for reproductive health services as a derived demand for reproductive health with adjustment costs. The theoretical predictions of the analysis are tested using individual level survey data from South Africa. The theoretical analysis suggests that the relationship between family planning services, their marginal costs, the stock of reproductive health capital and the woman's earnings volatility is essentially nonlinear; we find evidence in support of that hypothesis.

The third and final analysis chapter of the thesis is underpinned by research showing that ill-timed childbearing increases fertility and introduces a serious health hazard component into the human reproduction process, while also reducing economic opportunities available to women, preventing their long term economic prosperity and that of their children (see Gipson et al., 2008). This notion of 'ill-timing' requires some uncertainty. Even though it is commonly believed that contraception use gives women control over their fertility outcomes through the timing of births, the effectiveness of contraceptive use in controlling fertility remains a controversial issue and is still an under-researched question (see Cleland et al., 2006). Specifically, we argue that previous literature fails to capture fertility decisions about contraception efficiency and birth timing *as they are being made* (see Iyer and Velu, 2006), and ignores the central role that uncertainty plays in the human reproduction process (see Perrin and Sheps, 1964; Heckman and Willis, 1976). The model presented describes the way in which a woman's stock of reproductive health evolves over a birth interval, leading

[2]Sexual and reproductive health problems account for 18% of the total global burden of disease, and represent 32% of the burden among women of reproductive age (Singh et al., 2010).

up to the possible birth of a child. We find it to be a function of her contraception strategy and some random changes in her natural fecundability. We then apply a mixed hitting time (MHT) framework, assuming that a woman becomes pregnant when the underlying stochastic reproductive health process first crosses a threshold in zero (see Whitmore, 1979; Lee and Whitmore, 2006; Abbring, 2012), to estimate our model. As in the previous chapter, we find support for our theoretical analysis.

Chapter 2

The Economic Approach to Fertility: A Causal Mediation Analysis

2.1 Introduction

The large majority of contemporary economic fertility theories focus on the effects of parents' income and the opportunity cost of rearing children on completed family size. With a few exceptions (see Becker, 1960; Heckman and Willis, 1976; Michael and Willis, 1976), these theories fail to explicitly incorporate into the analysis inputs to the children-production function, such as fecundity and family planning services, despite the unquestionable role of these two factors in shaping a woman's fertility history. In particular, there is a growing body of evidence which suggests that falling national fertility levels since the 1990s are largely due to a rising proportion of demand for fertility control (see Caldwell et al., 1992; Johnson-Hanks, 2007; Moultrie et al.,

2012).

This study develops an integrated analysis of fertility choices (see Easterlin, 1975), where couples are both producing and utility maximizing units. In this model, a couple's capacity to procreate depends on their fecundity, as well as their contraception and sexual behaviours (see Becker, 1960; Heckman and Willis, 1976; Michael and Willis, 1976). At the same time, they are assumed to choose the ideal number of children by maximizing the utility of children, subject to a budget constraint reflecting the couple's income, and their specific explicit and implicit costs of rearing children. This approach is consistent with the literature on demographic transition, which postulates that the following three prerequisites should come to prevail for there to be a sustained fertility transition: (i) fertility must be within the calculus of conscious choice; (ii) effective techniques of fertility reduction must be accessible; and (iii) reduced fertility must be viewed as advantageous Coale (1984).

Moreover, one of the challenges to the current set of economic fertility theories is the ability to explain the negative fertility-income relationship regularly documented in empirical studies. Since the seminal paper by Becker (1960), much study has addressed the inconsistency between the empirical evidence of a negative fertility income effect and standard economic theory. Unfortunately, predictions in this regard, from most of the mainstream economic fertility models, are not robust and rely heavily on special assumptions regarding the functional form of the household's utility or production functions (see Jones et al., 2011).

Standard consumer choice models of household fertility postulate that children are normal goods. Consequently, one would expect that richer parents will have larger families than their poor counterparts. However, empirical evidence invariably suggests that, within a given society, fertility is often higher in poorer families (see Becker,

1960; Jones and Tertilt, 2008). This is also true across countries, where evidence suggests that countries with higher average fertility often have lower average levels of industrialization (see Galor and Zang, 1997; Bloom et al., 2009). As with any normal good, rising income would promote the increase in the quantity of children. However, explicit childrearing costs are socio-economic status expenditures directly related to parents' income, while implicit costs represent the opportunity cost of parent's time spent looking after a child, and are linked to wage rates in the labour market (see Becker, 1965; Mincer and Polachek, 1974). Both of these child costs would likely result in higher quality and a smaller number of children for richer families (see Becker and Lewis, 1973; Leibenstein, 1975; Caldwell, 1976).

Using a nonparametric causal mediation framework (see Pearl, 2009; Heckman and Pinto, 2013), our analysis explicitly explores the role of family planning services and the cost of children in mediating the causal effect of income on fertility, subject to unmeasurable fecundity and unobserved sexual risk taking behaviour. In particular, we decompose the total causal effect of income on fertility into a direct income effect, a price effect and a contraception effect. This qualitative causal model produces quantitative causal claims suggesting that the sign and magnitude of the causal relation between income and fertility depend on the relative sizes of the positive direct income effect, and the negative indirect effects mediated through the cost of childrearing and contraception. In this context, the total effect of income on fertility will be negative if the direct income effect is lower than the mediated effects, and positive otherwise.

Theoretically, our model is a modest contribution towards solving the ambiguity of the negative fertility-income relationship documented in various studies of the empirical population economics literature. In particular, the principal contribution of this study is to recast the *fertility-income* debate into the *income - cost of childrearing* -

contraception - fertility space, which effortlessly relates reproductive health outcomes to labour market outcomes. A pure reproductive health investment model suggests that investment in family planning is driven by a couple's desire to maximize their lifetime net money income, subject to the dynamics in their stock of reproductive health capital (see Ben-Porath, 1967). Thus, assuming a household money income production function (see Schultz, 1961; Ben-Porath, 1967; Mincer and Polachek, 1974), which varies with contraceptive efficiency in line with the human capital theory of demand for health (see Grossman, 1972, 2000; Becker, 2007), one should expect access to reproductive health services to influence participation or performance in the labour market. In our fertility model, fertility choices are linked to labour market outcomes through family planning and time allocation.

However, our causal mediation analysis of the economic approach to fertility is not only of theoretical interest. It also has significant policy implications in a variety of countries at different stages of the demographic transition. In the context of an aging population, it provides tools to evaluate family-friendly labour market policies aimed at boosting fertility, through the reduction of the cost of childrearing. Our analysis also provides a sound theoretical background for the evaluation of population policies aimed at reducing unwanted childbearing, through the subsidization of family planning services.

2.2 An Economic Fertility Model

2.2.1 Theoretical Background

The impact of income on fertility choices is not just statistical, it is inherently causal in nature. Understanding the interaction between biological and behavioural

determinants of fertility holds the key to understanding fertility trends in any society. In what follows, we present a number of factors, which are discussed in the literature as influencing fertility outcomes.

2.2.1.1 Fertility and Wages, Income and Wealth

There is robust empirical evidence suggesting that in most countries measures of fertility are negatively related to most measures of economic welfare most of the time. One approach in the main stream economics literature focuses on the significant role played by the opportunity cost of having a child as captured by wage rates. The latter are considered to be the shadow prices of time and highly correlated to income (Willis, 1973). In this context, the most common explanation for the negative fertility-income relationship is based on wage heterogeneity or differences in the tastes for quality of children. The basic notion being that, if childrearing is time intensive, the opportunity cost of time is very high for parents with higher wages. Thus, children will be more expensive for parents with higher wages, which should ultimately lead to fertility being negatively related to income (see Jones et al., 2011, for a review).

Starting with the work by Notestein (1945), contemporary work has also focused on the determinants of the *fertility transition*, as summarized in the evolution over time of the inverse relationship between a country's average fertility and the average income in the same country. A common finding in this literature is that the decline in fertility coincides with the onset of sustained per capita income growth.

Investigations interested in the Malthusian fertility cycles are among the oldest studies in the field of population economics. In his seminal work, Malthus (1993) predicted that fertility will remain at relatively high levels, such that rapid population growth will stifle improvements in the standard of living. However, this prediction,

so far, has failed to obtain; however, current versions of the Malthusian model have not, yet, reached a consensus around the direction of the relationship between fertility fluctuations and income. While some studies predict a positive correlation between income and fertility over the business cycle (Simon, 1969, 1977), others suggest that fertility may be counter-cyclical (see Butz and Ward, 1979).

In his wealth flow theory of fertility decline, Caldwell (1976) argues that the fundamental issue in demographic transition is the direction and magnitude of intergenerational wealth flows. This line of research postulates that a sustained per capita income growth produces a breakdown of the typical decision making structure, which leads to a reversal of wealth flows within the family. When the flow of money, goods, and resources is from the younger to the older generation, an increase in wealth will result in higher fertility rates. But when the flow of wealth is from parents to children, being childless is the most rational economic behaviour, since the degree of altruism toward each child varies inversely with the number of children (see Becker and Barro, 1988). In turn, this reversal, which is mainly expressed by the individualistic desires of parents to promote the health and education of their children, makes children more costly to parents, ultimately leading to a decline in fertility (Caldwell, 1982). This is also consistent with the economic fertility theory developed by Leibenstein (1975), which predicts a negative fertility-income link using the concept of status expenditures.

In agrarian societies, differentials in fertility by household land ownership status is more pronounced than by income level, and contain information about optimal reproductive behaviour based on differential demand for children and the services they provide to parents. In particular, land appears to have at least two distinct aspects that influence fertility in opposite directions. One is the size of land a household

controls for cultivation purposes, including leased land and excluding owned land rented out. A second dimension is land ownership, which refers to the system of distribution of land and produce from the land (Cain, 1985).

Furthermore, it has been suggested that the size of the area cultivated by a family affects fertility by modifying the relative cost of an extra child. In this case, families with access to larger holdings are able to use additional family labour more profitably, which leads to higher fertility; the complementarity of child labour and landholdings underlies the positive relationship between farm size and fertility. On the other hand, ownership and operation of land not only entails returns to labour and management accruing to cultivators of rented land, it also provides an equity return. Consequently, land ownership should reduce the importance of children as sources of parental security in old age and contribute to lower fertility. Thus, the negative relationship between land owned and fertility (Cain, 1985; Stokes et al., 1986).

2.2.1.2 Fertility and Education

Almost every discussion of fertility differentials makes reference to the role of education in reproductive decisions. A number of studies have used education as a proxy of lifetime income, making the literature on the interaction between education and fertility similar in many ways to that on income and fertility (Jones and Tertilt, 2008). From a theoretical standpoint, the role of education in fertility decline has been analyzed with reference to Coale1984 preconditions for fertility transition mentioned earlier. In particular, the influence of education is closely related to the first two prerequisites that concern the individual's ability to control the human reproductive process (Cleland and Wilson, 1987). The main idea being that the growth in formal education brought about by the structural modernization of societies has created a

psychological shift empowering individuals with a sense of control over their own destiny and the ability to follow their own pursuits. Such a general shift in outlook would change individual's attitudes towards and increase the propensity to use birth control.

There is overwhelming evidence to support the notion that, during transition, high levels of parental education are associated with lower marital fertility through greater family planning service use. In particular, borrowing from the literature on the relationship between mother's education and infant mortality, more recent studies have found that, not only does the education of the mother exert a strong influence on infant mortality (Caldwell, 1979), it appears to be a very strong predictor of the decline in fertility, as well (see Schultz, 1986; Preston and Hartnett, 2010). This link tends to be relatively stronger compared to other purely economic covariates, such as income and occupational status. In most countries, a few years of schooling are sufficient for a shift in reproductive behaviour through changes in perceptions, ideas and aspirations (Cleland and Wilson, 1987). Thus, the prediction that, as countries adopt universal primary schooling, fertility would eventually fall, naturally (Caldwell, 1982).

The literature also does differentiate between the fertility impact of the level of education received by parents themselves, from the influence of schooling opportunities of their offspring. The growth in educational opportunities for children encourages investment in child quality rather than quantity (see Becker and Lewis, 1973). If children are indeed normal goods, then quantity should change directly with income. However, in Becker's *quantity-quality* model of fertility choice, a parent has to decide between having a large number of children with few economic opportunities each, or having few children with a large bequest (e.g. receiving a large investment in educa-

tion). Thus, it is not surprising if, at the level of the individual family, aspirations for child education are often found to be negatively correlated with overall family size desires (see Cleland and Wilson, 1987; Jones and Tertilt, 2008).

2.2.1.3 Fertility and Family Planning

A few empirical studies have evaluated the role of family planning in inducing a decline in fertility rates. In particular, there is growing evidence to suggest that much of the fertility decline in sub-Saharan Africa is due to widespread contraceptive use (Caldwell et al., 1992; Timaeus and Moultrie, 2008). Research using data from the US also suggests that the introduction of family planning was associated with significant and persistent reductions in fertility, driven both by falling completed childbearing and childbearing delay (Kearney and Levine, 2009; Bailey, 2012).

The demographic rationale for family planning is to avoid pregnancy, or at least reduce the risk of conception associated with sexual intercourse, through reduction in fecundity (Moultrie et al., 2012). However, rather than explicitly analyzing the causal effects of contraception on fertility, the majority of theoretical studies in the literature are concerned with the determinants of contraceptive use. Much of the analysis, in both the fertility decline literature (Coale, 1984; Cleland and Wilson, 1987) and socio-economic theory of fertility (Easterlin, 1975; Bongaarts, 1993), is focused on the impact of socio-cultural factors and the opportunity costs of family planning services on the demand for contraception. In particular, despite the fact that earlier work on fertility choice contains a discussion on the impact of differential contraceptive knowledge (see Becker, 1960; Schultz, 1973), the role of contraception efficiency in reducing fecundity has received much less attention in both the theoretical and empirical literatures.

The dominant model in the socio-economic literature, and is among the first to explicitly incorporate contraception into the fertility choice analysis, follows a supply-demand approach to fertility (see Easterlin, 1975; Easterlin et al., 1988; Bongaarts, 1993). This framework postulates that the determinants of fertility and contraception work through at least one of the following: (i) *The demand for children*, which should correspond to the desired family size; (ii) *The supply of children*, taken to correspond to the number of surviving children parents would have without controlling their level of fecundity; and (iii) *The opportunity and psychic costs* of birth control. On one side, the demand for children varies with factors influencing fertility choices via their effect on household income and the price of children. On the other side, the supply of children is a function of the parent's natural fertility and the survival rate of newborns to adulthood (Easterlin et al., 1988). Unfortunately, in this framework the causal path between contraceptive efficiency and fecundity is not made clear, and the implicit assumption of perfect control over the probability of getting pregnant makes contraceptive efficiency redundant in the modeling exercise.

A very limited number of studies have also explicitly incorporated contraceptive efficiency in the theoretical analysis of fertility choices (see Heckman and Willis, 1976; Michael and Willis, 1976). Using a household production model, this strand of the literature assumes imperfect contraception, so that a couple's natural ability to procreate is altered by the efficiency of their contraceptive strategy. This approach has the advantage of taking into account the impact of opportunity costs on the demand for family planning, while explicitly establishing the causal path between contraception use and fertility. In this setting, a couple chooses the optimal level of contraceptive efficiency as to equate the marginal benefit and marginal cost of contraception. The marginal benefit of contraception, or its continuation value, is

defined as the expected utility of preventing a conception (Heckman and Willis, 1976). Also, the sign and magnitude of the continuation value of contraception plays a pivotal role in explaining the impact of economic variables such as labour market outcomes on contraception strategies and realized fertility.

2.2.1.4 Fertility and Fecundity

It is difficult to assume that differences in fertility rates all over the world would be explained by behavioural factors alone. Apart from behavioural factors, such as contraceptive use and sexual behaviour, fertility differentials might also be an indication of some latent differences in biological factors, such as human fecundity (Gini, 1926; Skakkebaek et al., 2006; Louis, 2011).

In the literature, fecundity is generally defined as the biologic capacity for reproduction during a normal menstrual cycle with sexual relations and no contraception (Gini, 1926; Leridon, 2007). As such, fecundity outcomes are couple dependent and are a function of the timing and frequency of sexual intercourse along with the biological reproductive capacity of the two partners. While fertility, measured by births, demonstrates fecundity, the latter is directly linked to and is generally measured by the concept of time to pregnancy. The time to pregnancy, thus, is the probability of conception for women exposed to unprotected sexual intercourse in the absence of lactational anovulation, pregnancy, or sterility (see Louis, 2011).

Furthermore, researchers have long focused on female fecundity. But recently, interest has arisen around the issue of male fecundity, given the growing body of evidence suggesting that male fecundity may be on the decline, due to modern lifestyle choices and exposure to environmental factors harmful to sperm production; all of these potentially decrease the odds of fertility. In particular, Skakkebaek et al. (2006)

argue that exposure to adverse biological factors, such as endocrine disruptors causing a deterioration in male reproductive health, may be a contributing factor to the recent decline in fertility rates in many industrialized countries. Endocrine disruptors are the root causes of male reproductive health problems, such as testicular cancer, undescended testicles and poor semen quality. A series of published studies, using data from men around the world with no history of infertility, provides evidence to suspect an overall reduction in male fertility, due to poor quality semen (see Carlsen et al., 1992; Skakkebaek et al., 2006).

Lastly, incorporating human fecundity in a fertility model requires recognition of the many methodological challenges stemming from the latent nature of fecundity and the couple-dependent characteristics of its outcomes. One of the main challenges is that fecundity is not a dichotomy, but a continuum whose value is highest in the first few months that couples are attempting to conceive, relative to later months (see Tietze, 1959). This pattern suggests that couples differ considerably in their ability to achieve pregnancy, which precludes the conceptualization of individuals or couples by fecundity status and implies non availability of suitable biomarkers for population-based research (Louis, 2011). In what follows, our fertility model treats human fecundity as an unobservable covariate influencing both a woman's contraceptive behaviour and her fertility outcomes.

2.3 The Causal Path to Conception or Birth

In what follows, we formalize the causal link between endogenous labour and reproductive health outcomes using a fertility model based on a woman's money income production function. In the end, we illustrate all the causal mechanisms using a causal path diagram (Pearl, 2009).

Assume that any fecundable woman i is naturally endowed with a vector of unobserved abilities U_i, which consists of both cognitive and noncognitive skills affecting her schooling decisions, as well as her labour and behavioural outcomes (see Heckman, Stixrud and Urzua, 2006). In particular, cognitive abilities, such as intelligence, indirectly affect the woman's probability to secure a wage rate W_i in the labour market through observed schooling decisions S_i; noncognitive traits, such as motivation and persistence, directly determine W_i. Thus, the woman's wage rate function is given by

$$W_i = W_i(S_i, U_i, \varepsilon_{iw}), \tag{2.1}$$

where ε_{ia} is the idiosyncratic error term related to the variable a.

We assume that the demand for family planning is underpinned by investments in reproductive health capital. Using the human capital theory of the demand for health (Grossman, 1972, 2000; Becker, 2007), we suppose that contraception increases the time a woman remains active in the labour market. Therefore, the time spent in the labour market, H_i, can be assumed to vary with the levels of family planning, Z_i, and childcare services, N_i, available to the working mother

$$H_i = H_i(N_i, Z_i, \varepsilon_{ih}). \tag{2.2}$$

The number of hours spent in non-childrearing production activities is the main input in the money income production technology. Thus, Equations (2.1) and (2.2) imply the following equation for the woman's money income production function

$$Y_i = Y_i\Big(H_i, W_i, \varepsilon_{iy}\Big) - K_i(N_i, Z_i, \varepsilon_{ik}), \tag{2.3}$$

where ε_{iy} is the error term which might include other sources of income, and K_i is the cost involved in participating in the labour market, such as the payment for family planning services, the services of a nanny or commuting to work. The included wage rate represents the time cost of children (see Mincer and Polachek, 1974).

We also make provision for the growth in income to shift the household into a higher socio-economic status, which translates into higher expenses per child, and implies a direct relationship between income and the price of children (see Leibenstein, 1975; Caldwell, 1976). In this context, the cost of childrearing X_i combines both expenditures on children-related products, which varies with income, and the opportunity cost of parental time measured by the wage rate

$$X_i = X_i\Big(Y_i\big(H_i, W_i, \varepsilon_{iy}\big) - K_i(N_i, Z_i, \varepsilon_{ik}), W_i(S_i, U_i, \varepsilon_{iw})\Big). \tag{2.4}$$

We also assume that a couple is a production unit. In the biological reproduction process, latent abilities have a direct impact on contraception behaviour and indirectly through education. Thus, a woman's demand for contraception efficiency is given by

$$Z_i = Z_i(S_i, Y_i, U_i, V_i, \varepsilon_{iz}), \tag{2.5}$$

where V_i is latent fecundity, which captures uncertainty in the human reproduction process and is a function of a variety of unobserved biological factors, including the semsn quality of the woman's partner. For example, an infertile woman or a fecundable woman who is experiencing a temporary sterile period for a variety of reasons, such as lactational amenorrhea, is not likely to use contraception during intercourse.

It is clear that each variable a in the model is generated by a nonparametric structural equation that relates a variable to its immediate causes, and to its idiosyncratic

error, ε_a – we have dropped the i subscript to simplify the notation – via some arbitrary deterministic function. All the nonparametric functions are invariant under external manipulations of their arguments (Frisch, 1938), and the error terms are mutually independent, $\varepsilon_a \perp\!\!\!\perp \varepsilon_b,\ a \neq b$. The random nature of these error terms implies that our fertility model is also stochastic.

To simplify the analysis, we assume, without loss of generality, that the cost function $K_i(\cdot)$ is zero and that the level of childcare services is exogenous. Then, keeping implicit the error terms and all other exogenous variables, the causal assumptions conveyed by our fertility model translate into the following fertility production function

$$Q = f\Big[Y\Big(H(Z), W(S,U)\Big), X\Big(Y\Big(W(S,U)\Big), W(S,U)\Big), Z\Big(S, Y\Big(W(S,U)\Big), U, V\Big), v\Big]. \quad (2.6)$$

The diagrammatic description of this nonparametric structural equation fertility model is given in Figure 2.1. In particular, this cyclical causal model depicts the mediation structure of a model, where some of the time lags are assumed to approach zero (see Fisher, 1970).

For the purpose of this study, we use a version of Figure 2.1 which assumes no simultaneity and abstract from the feedback effect of birth control on income, as mediated by the time spent in the labour market. This simplification produces the directed acyclical graph (DAG) in Figure 2.2, where no variable is a descendant of itself. The model now consists of eight random variables: Y stands for the level income a woman receives, Z is the efficiency of the contraceptive strategy a woman follows, Q represents a woman's fertility outcome, S stands for the highest level of

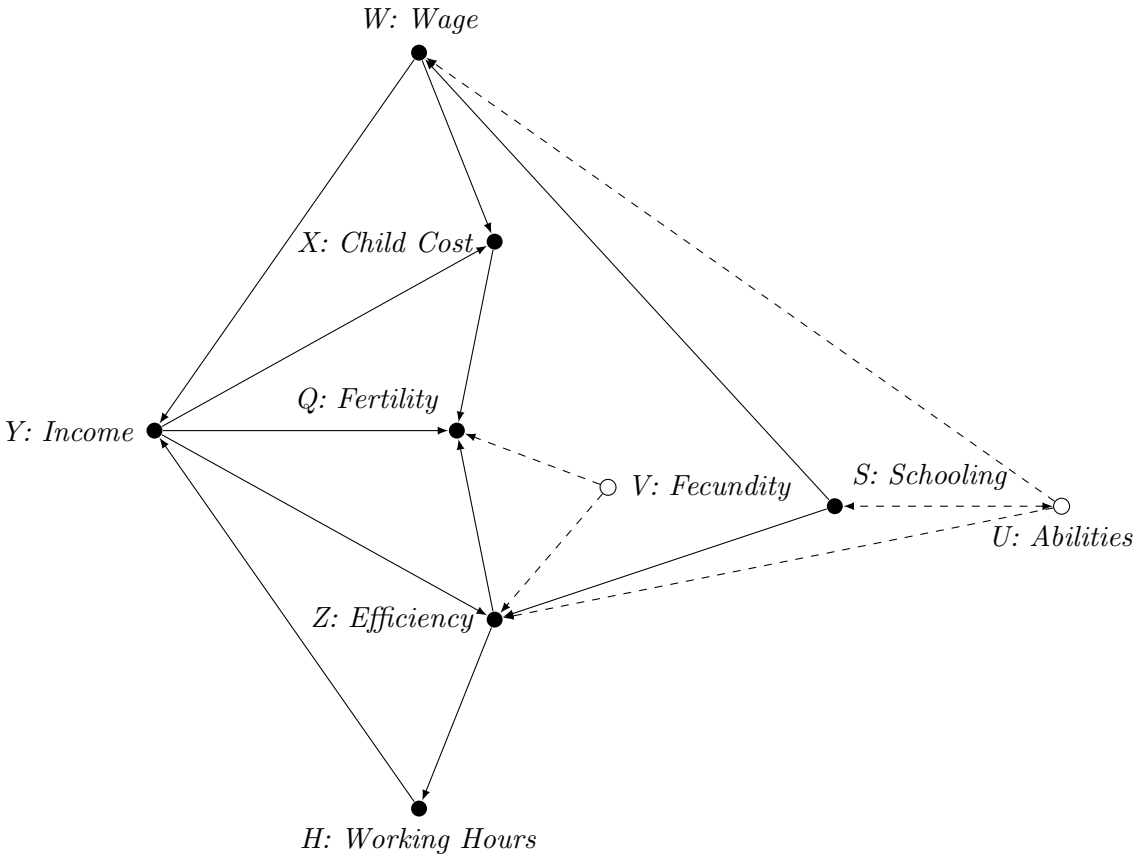

Figure 2.1: The Cyclical Causal Fertility Model. Solid bullets represent observed variables, hollow circles represent unobserved (or latent) variables.

education achieved by a woman, W is the wage rate, X stands for the per capita cost of childrearing, latent variables U and V represent skills and fecundity, respectively.

Keeping implicit the error terms, the structural equations for the simplified structural model translate into the following fertility production function

$$q = f\Big[y\Big(w(s,u)\Big), x\Big(y\Big(w(s,u)\Big), w(s,u)\Big), z\Big(s, y\Big(w(s,u)\Big), u, v\Big), v\Big]. \tag{2.7}$$

Under appropriate properties of continuity and derivability, the total effect of income

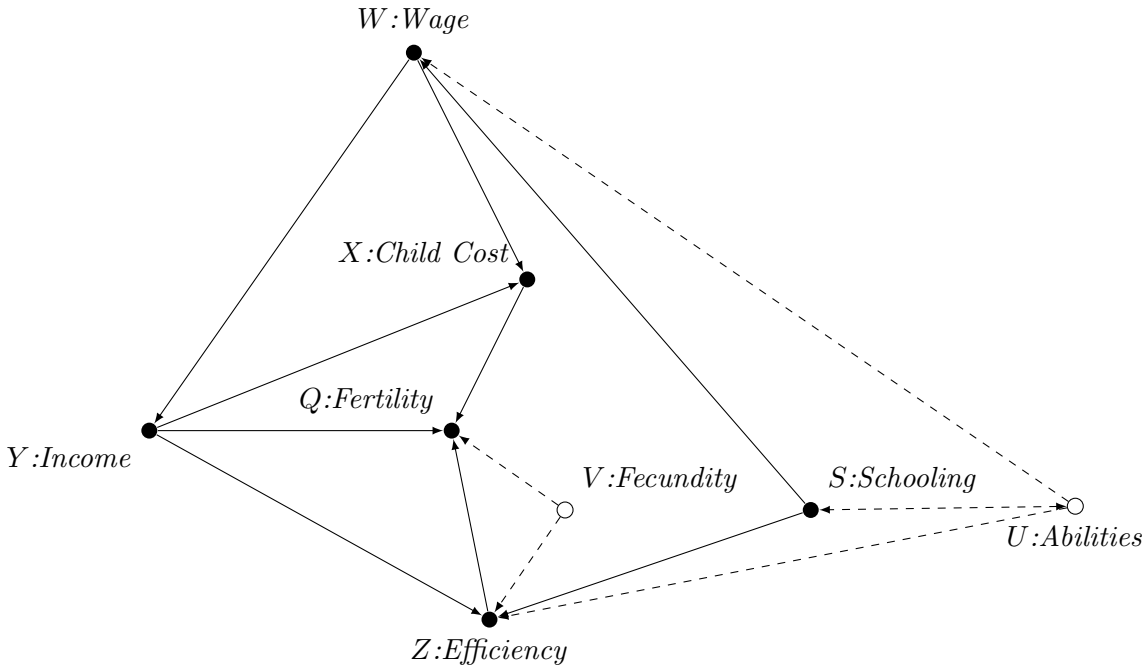

Figure 2.2: The Acyclical Causal Fertility Model. Solid bullets represent observed variables, hollow circles represent unobserved (or latent) variables.

on fertility related to the model in Equation (2.7) is given by

$$\underbrace{\frac{dq}{dy}}_{\delta:\ Total\ Causal\ \text{Effect}} = \underbrace{\frac{\partial f(\cdots)}{\partial y}}_{\delta_y:\ Pure\ Income\ \text{Effect}} + \underbrace{\frac{\partial f(\cdots)}{\partial x}\frac{\partial x(\cdots)}{\partial y}}_{\delta_x:Price\ \text{Effect}} + \underbrace{\frac{\partial f(\cdots)}{\partial z}\frac{\partial z(\cdots)}{\partial y}}_{\delta_z:Contraceptive\ \text{Effect}} \tag{2.8}$$

where δ is the *total effect* of income on fertility measured as the effect of change in income, combined with the change in fertility mediated by the change in contraceptive efficiency or the cost of childrearing; the income effect δ_y captures the *direct effect* of the change in income for a given level of contraceptive efficiency and cost of children; the price effect δ_x is the *indirect effect* operating through the opportunity cost of children; the contraceptive effect δ_z is also an indirect effect of income via the demand for family planning services.

The sign of the different effects represents a stable economic relationship that only

depends on the economic nature of children, and is transportable across any cultural and sociological environments that may affect fertility outcomes. The direction of the total effect in Equation (2.8) would depend on the sign and the relative sizes of the direct income, price and contraception effect, respectively. The total effect of income on fertility will be positive if the direct income effect is greater than the mediated price and contraceptive effects combined, and negative if the opposite is true. Note that the magnitude of the total effect is also affected in the same fashion, despite the fact that the total effect may be weak or nonexistent, due to the cancellation of effects. This would be the case when a strong positive direct effect is canceled by equally strong negative mediated effects. Thus, it is possible for the total effect to be weak or nonexistent, while direct and indirect effects are both strong.

The direct income effect, δ_y, would always be positive – if we assume that children are normal goods, children are an increasing function of income (i.e. richer parents would naturally have larger families). The price effect, δ_x, is the indirect effect of income on fertility mediated by factors indicative of the cost of having children. These factors include the opportunity cost of time, as measured by the wage rate (see Mincer and Polachek, 1974), and the social-class related expenditures on children, which are assumed to be positively related to the family's income (see Martineau, 1958; Hotz et al., 1997). It is clear that, by the Law of Demand, an increase in the cost of children will decrease the demand for children. Lastly, the contraceptive effect, δ_z, describes the impact of a change in income on the supply of children, via the demand for family planning. Assuming contraceptive efficiency is a normal good, higher earning couples will tend to use more efficient family planning services, which will results in a smaller completed family size, *ceteris paribus*.

2.4 Econometric Analysis

This section reviews the conditions necessary for the identification and estimation of the causal effects of income on fertility outcomes, from the perspective of a non-parametric structural equation framework (see Petersen et al., 2006; VanderWeele, 2009; Pearl, 2009, 2012*b*). We use contraceptive efficiency and the cost of childbearing as mediator variables. In particular, we discuss the conditions under which the direct income, price and contraceptive effects, defined in the previous section, can be identified and estimated using observational studies.

2.4.1 The Counterfactual Definition of Causal Effects

Causality is essentially a thought experiment (Frisch, 1930; Haavelmo, 1943, 1944). In the counterfactual framework, for example, the causal effect of treatment on an individual is defined as the difference in outcome if the same individual was exposed to a treatment versus unexposed (Rubin, 1974; Holland, 1986). However, the fundamental problem of causal inference implies that one can never find the true causal effect, since it is impossible to observe the value of the outcome under both treatment and no treatment on the same individual (Holland, 1986).

This hypothetical nature of the causal analysis implies, in the context of structural causal inference (see Pearl, 2009), that the various effects described in Equation (2.8) would have causal meanings if and only if we had a way of delivering counterfactual information by hypothetically manipulating the causal paths implied by the system of equations in Equation (2.7). Following the work by Frisch (1930, 1938) and Haavelmo (1943, 1944), the mental manipulation of causal mechanisms mainly involves hypothetical variations in treatment levels, in a way that does not affect the values of other variables in the model. In practice, the mental variation of a causal path often

entails variation in hypothetical exogenous inputs (see Heckman and Pinto, 2013), and hypothetical disablement of specific direct causal links (see Pearl, 1995).

Using the hypothetical approach to causality, we can define a range of causal effects as discussed in the mediation analysis literature (see Pearl, 2012*b*). These effects include the *total effect*, (TE), the *natural direct effect*, (NDE), and the *natural indirect effect*, (NIE). Although similar counterfactuals have been defined before in other fields of research, our analysis is a comprehensive application of a general counterfactual framework to fertility choices that integrates the utility maximization and production functions of the household. The framework is flexible enough to simultaneously accommodate several aspects of the economic approach to fertility, and can be used to evaluate a wide range of family policies.

The TE is the impact on fertility resulting from a move in income from y to y', with the mediator variables X and Z being allowed to track the change in Y as dictated by the model in Equation (2.7)

$$\begin{aligned} TE &= E\{f[y', x(y', w(s,u)), z(s, y', u, v), v] - f[y, x(y, w(s,u)), z(s, y, u, v), v]\} \\ &= E\{Q(y')\} - E\{Q(y)\} \end{aligned} \tag{2.9}$$

where $Q(y)$ denotes the outcome Q when treatment Y is fixed at y.

The *natural direct effect* (NDE) is defined as the expected change in outcome, induced by moving treatment from y to y' while setting the intermediate variables to whatever value they would have attained in the absence of treatment. This definition implies that one must find a way to disable the ability of mediating variables to respond to treatment, while allowing them to vary with the other individual characteristics in the model, as if the treatment did not change. In our model, this counterfactual definition translates into the following expression for the Direct Income Effect

(DIE)

$$\begin{aligned} DIE &= E\{f[y', x(y), z(s, y, u, v), v] - f[y, x(y), z(s, y, u, v), v]\} \\ &= E\{Q(y', X_y, Z_y)\} - E\{Q(y, X_y, Z_y)\}. \end{aligned} \tag{2.10}$$

Thus, the DIE is defined as the expected effect of income on fertility outcomes while blocking the impact of increases in income on the demand for contraceptive efficiency or the price of children. In other words, the DIE measures the expected impact of income on fertility, while setting the demand for contraceptive efficiency and the cost of childrearing for each individual to whatever value they would have attained prior to the change in income. This counterfactual definition of the DIE suggests that, other things being equal, growth in family income would have a positive impact on completed family size.

In the same vein, the hypothetical model can be used to define the *natural indirect effect* (NIE) of treatment as the portion of total effect explained by the mediating variables, if the outcome had not responded to changes in treatment. It follows that the definition of the indirect effect involves nested counterfactuals, since it requires fixing the treatment itself, at its reference level, while allowing the mediators to change to new levels, the ones they could have attained under treatment (see Pearl, 2001; Petersen et al., 2006). In our case, we have two types of NIEs: the Price Effect (PE) and the Contraception Effect (CE).

The expression for CE is given by

$$\begin{aligned} CE &= E\{f[y, x(y), z(s, y', u, v), v] - f[y, x(y), z(s, y, u, v), v]\} \\ &= E\{Q(y, X_y, Z_{y'})\} - E\{Q(y, X_y, Z_y)\}. \end{aligned} \tag{2.11}$$

The magnitude of the CE is of particular interest for the evaluation of family planning programs aimed at reducing unintended childbearing by subsidizing contraception. However, the question of how family planning programs have shaped family size remains controversial (Bailey, 2012). Some economic models predict that family planning programs could reduce childbearing (see Becker and Lewis, 1973; Michael and Willis, 1976; Kearney and Levine, 2009), while others predict quite the opposite (see Ananat et al., 2009). As a basis for analysis, it is natural to assume that contraception is linked to fertility through its impact on the probability of getting pregnant through the following relationship (Heckman and Willis, 1976):

$$p = (1 - z)v, \quad 0 < p, z, v < 1, \tag{2.12}$$

where p is the probability of falling pregnant, z is contraception efficiency, and v is fecundity. Assuming contraception efficiency is a normal good, then the CE in Equation (2.11) implicitly recognizes an inverse link between contraception use and birth interval length (see Yeakey et al., 2009), which ultimately results in a negative fertility response to a rise in contraception efficiency levels, consistent, in our case, to an increase in income from y to y'.

In the same fashion, the expression for the PE reads as

$$\begin{aligned} PE &= E\left\{f[y, x(y'), z(s, y, u, v), v] - f[y, x(y), z(s, y, u, v), v]\right\} \\ &= E\left\{Q(y, X_{y'}, Z_y)\right\} - E\left\{Q(y, X_y, Z_y)\right\}. \end{aligned} \tag{2.13}$$

The identification and estimation of the PE is not only of theoretical interest; it also has significant policy implications in countries with an aging population; decreased fertility and increased life expectancy has aged the population of a number

of developed countries, posing a long term risk to economic growth and a threat to the sustainability of the welfare state. This decline in fertility, which has induced a below-replacement fertility rate in these countries, is being blamed partially on the increased cost of raising children owing to economic development (see Kalwij, 2010). A variety of family-friendly labor market policies, such as family allowances, child-care subsidies, and maternity-parental leave benefits, have been proposed in order to create opportunities for women to reconcile family life and employment. Using our model, one can evaluate the fertility response of these employment-oriented family policies by estimating the PE as defined in Equation (2.13). In this context, the PE captures a key prediction of our fertility model: that fertility will respond to changes in the price of children.

2.4.2 Identification

In the analysis of causal effects, identification seeks to answer the fundamental question of whether, given a set of assumptions, the modified distribution required for the definition of counterfactuals can be estimated from observational data. Thus, in this context, the goal of the identification procedure is to express the causal parameters from the *hypothetical* model using observed probabilities of the *empirical* model that governs the data-generating process (see Pearl, 2001, 2009; Heckman and Pinto, 2013). For example, given two random variables (X, Y), identification may concern whether the hypothetical query $Q = P(Y|X \ fixed \ at \ x) = P(Y(x))$ is identifiable from the empirical model $P(X, Y)$ and, if so, how can it be estimated? More generally, identification requires the move from the causal assumptions encoded in a hypothetical causal model to associations observable in the data.

Consider a model that describes the causal mechanisms among a set of vari-

ables $\mathcal{L} = \{K_1, \cdots, K_n\}$ associated with a set of mutually independent error terms $\boldsymbol{\varepsilon} = \{\varepsilon_1, \cdots, \varepsilon_n\}$ through a system of autonomous structural equations $\boldsymbol{g} = \{g_1, \cdots, g_n\}$. We are interested in the causal relationships $K_i = g_i(Pa(K_i), \varepsilon_i)$, where the *endogenous* variable K_i is directly caused by its parents $Pa(K_i) \subset \mathcal{L}$ and $\varepsilon_i \in \varepsilon$. Those variables directly or indirectly causing K_i are called its ancestors $An(K_i) = \{K_j \in \mathcal{L}; K_i \in De(K_i)\}$. Similarly, $De(K_i) = \{K_i' \in \mathcal{L}; K_i \in An(K_i')\}$ are all descendant variables directly or indirectly caused by a variable K_i. If $Pa(K_i) = \varnothing$ then K_i is an *exogenous* variable determined outside the model. By analogy, a set of variables that have K_i as a parent are called children of K_i, $Ch(K_i) = \{K_i' \in \mathcal{L}; K_i \in Pa(K_i')\}$.

2.4.2.1 Conditional Independence

The statistical information encoded in a causal model is completely captured by conditional independence relationships among the variables in the model. Consider a DAG which contains n variables, $\{K_1, \cdots, K_n\} = \mathcal{L}$. The local Markov property states that, under mutually independent error terms, each variable in a recursive model is conditionally independent of its non-descendants given its parents:

$$K \perp\!\!\!\perp \mathcal{L} \backslash [De(K) \cup Pa(K)] | Pa(K). \tag{2.14}$$

If the n variables in the model form a joint probability distribution $P(k_1, \cdots, k_n)$, then this property is equivalent to the following Markov factorization property:

$$P(k_1, \cdots, k_n) = \prod_{i=1}^{n} p(k_i | Pa(k_i)), \tag{2.15}$$

which in turn is equivalent to the global Markov property as defined by the concept of directional separation, or d-separation in short (Pearl, 1988). The concept of

d-separation is a graphical criteria, which allows researchers to read all structural implications of a causal model from their respective diagrams.

(*d-separation*): A path p is blocked by a conditioning set of variables B if either (1) p contains a *chain* $i \to m \to j$ or a *fork* $i \leftarrow m \to j$, such that (the middle variable) $m \in B$; or (2) p contains a *collider* $i \to m \leftarrow j$, such that neither the middle variable m, nor any descendant of m, is in B. If B blocks *all* paths from set X to set Y, it is said to "*d-separate* X and Y".

The concept of d-separation gives rise to the d-separation theorem widely used in nonparametric causal mediation analysis (Verma and Pearl, 1991; Pearl, 2009).

Theorem 2.4.1. d-separation *theorem: If if two sets of variables* X *and* Y *are d-separated by another set of variables* B *along all paths in a graphical causal model, then* X *is* statistically independent *of* Y *conditional on* B *in every distribution compatible with the graphical causal model:* $X \perp\!\!\!\perp Y|B$.

The empirical content of different causal effects can be identified by considering meaningful interventions on specific variables of interest. In our approach, a different type of causal effect gives rise to a structurally different hypothetical model, characterized by a different set of pairwise marginal and conditional independencies. In what follows, we use the d-separation criteria to reveal the structure of associations and independencies, which could be observed if the data were generated according to the causal assumptions encoded in each hypothetical causal fertility model. In this study, we assume that all testable implications are derived from the hypothetical manipulation of the causal graph in Figure 2.2.

Consider an intervention $do(k)$ that fixes the value of a set of variables K to some constant value k, irrespective of the usual behaviour of K given by the empirical joint distribution $P(\mathcal{L})$. Every such intervention in a statistical model would con-

tain testable causal implications, only if the resulting 'interventional' distribution is expressible in terms of conditional distributions:

$$P(\mathcal{L}\backslash K_j|\text{do}(k)) = \prod_{k_i \notin K_j} P(k_i|Pa(k_i)). \tag{2.16}$$

where the local Markov assumption enables the move from the causal assumptions encoded in a hypothetical causal model to associations observable in the data. Equation (2.16) is the so-called truncation formula (Pearl, 2009), where the intervention has removed all $P(k_j|Pa(k_j))$ terms from the Markov factorization, such that the parents of every variable are that variable's direct causes.

In order to operationalize hypothetical manipulations of the causal relationships required by the counterfactual definition of the various causal effects, we expand the empirical model with an hypothetical treatment variable $\tilde{K}_j$ related to the treatment variable K_j. For consistency, we assume that assigning a value k to $\tilde{K}_j$ is equivalent to observing K_j attain a value k. (The *hypothetical* variable): We define an hypothetical variable $\tilde{K}_j$, associated with the treatment variable K_j, such that

(1) $An(\tilde{K}_j) = \varnothing$,

(2) $\tilde{K}_j \in Pa(K_i)$, and

(3) $[De(\tilde{K}_j) \in \tilde{\mathcal{L}}] \subseteq [De(K_j) \in \mathcal{L}]$,

where K_i is any variable in the empirical model with parent K_j, and the lists of variables in the empirical and hypothetical models are $\mathcal{L}$ and $\tilde{\mathcal{L}} = \mathcal{L} \cup \{\tilde{K}_j\}$, respectively.

We assume that the hypothetical model is equivalent to the empirical model, in the sense that they both share common features and encode the same sets of conditional independence relations (see Verma and Pearl, 1991; Ali et al., 2009). In order to preserve equivalence, the general necessary condition is that the hypothetical

manipulation should not create or destroy any independence in the modified model (Pearl, 2012*a*). In other words, the hypothetical variable should leave dependent variables dependent, and those variables that are independent in the observed model, may not turn into dependent variables in the hypothetical model. the fact that the new variable $\tilde{K}_j$ is to replace, in the hypothetical model, all K_j-input of at least one variable in $Ch(K_j)$ should not change the character of the conditional distribution of the other variables in the target causal graph.

The concept of d-separation can also be used as a test of whether a given hypothetical manipulation of the empirical variable is an equivalence preserving modification. A hypothetical modification of the data generating process maintains equivalence if it neither creates nor destroys any d-separation condition in the resulting hypothetical model (Verma and Pearl, 1991; Pearl, 2009). In our analysis, equivalence is preserved through a requirement of common support for $(K_j, \tilde{K}_j)$, invariant structural equations and stationary distribution of error terms ε. These conditions ensure that the distribution of any variable conditional on K_j being equal to $\tilde{K}_j$ in the hypothetical model is equal to the distribution of that specific variable in the data generating process. The following theorem links the probability measures of our hypothetical and empirical fertility models, and shows that these two causal models are equivalent (Verma and Pearl, 1991; Ali et al., 2009):

Theorem 2.4.2. *Let $\tilde{K}_j \in \tilde{\mathcal{L}}$ be the hypothetical variable in the hypothetical model associated with treatment variable K_j. Assume $\tilde{K}_j$ is uniformly distributed in the support of treatment variable K_j. Then, the empirical and hypothetical models define the same probability distribution $P(\tilde{\mathcal{L}}|K_j = \tilde{K}_j) = P(\mathcal{L})$.*

Proof.

$$
\begin{aligned}
&P(\tilde{\mathcal{L}}|K_j = \tilde{K}_j) \\
&\quad = P(\mathcal{L} \cup \tilde{K}_j | K_j = \tilde{K}_j) \\
&\quad = P(\mathcal{L}|K_j = \tilde{K}_j) + P(\tilde{K}_j|K_j = \tilde{K}_j) - P(\mathcal{L} \cap \tilde{K}_j|K_j = \tilde{K}_j) \\
&\quad = \sum_{k \epsilon supp(K_j)} P(\mathcal{L}|K_j = k, \tilde{K}_j = k) \frac{P(K_j = k, \tilde{K}_j = k)}{\sum_{k \epsilon supp(K_j)} P(K_j = k, \tilde{K}_j = k)} \\
&\quad = \sum_{k \epsilon supp(K_j)} P(\mathcal{L}|K_j = k, \tilde{K}_j = k) \frac{P(K_j = k) P(\tilde{K}_j = k)}{P(\tilde{K}_j = k) \sum_{k \epsilon supp(K_j)} P(K_j = k)} \\
&\quad = \sum_{k \epsilon supp(K_j)} P(\mathcal{L}|K_j = k, \tilde{K}_j = k) P(K_j = k) \\
&\quad = \sum_{k \epsilon supp(K_j)} P(\mathcal{L}|K_j = k) P(K_j = k) \\
&\quad = P(\mathcal{L}).
\end{aligned}
$$

The second equality follows an application of the inclusion-exclusion principle. The third equality stems from the fact that $P(\mathcal{L} \cap \tilde{K}_j|K_j = \tilde{K}_j) = P(\tilde{K}_j|K_j = \tilde{K}_j)$. The fourth equality comes from the local Markov property in Equation (2.14), since $\tilde{K}_j \notin De(K_j)$. The fifth equality results from the fact that $\sum_{k \epsilon supp(K_j)} P(K_j = k) = 1$ and the assumption that the hypothetical variable $\tilde{K}_j$ is uniformly distributed, which suggests that $P(\tilde{K}_j = k) = c$, a constant. The sixth equality originates from the so called consistency assumption that guided the definition of the hypothetical variable. □

The equivalence between the hypothetical and the empirical distributions implies that the causal effects defined in the hypothetical model can be identified using data generated by the empirical model. In particular, equivalence between the two models provides an opportunity to identify a number of causal effects in the model by replacing at least one disabled causal link originating from K_j, with a corresponding

hypothetical $\tilde{K}_j$-input, and then evaluate the impact of this hypothetical modification on the conditional probabilities in the resulting hypothetical model. It follows that, by the Markov factorization property, the factorization of the joint distribution of the variables in the set $\tilde{\mathcal{L}}$ of the hypothetical model yields

$$P(\tilde{\mathcal{L}}\backslash\{\tilde{K}_j\}|\tilde{K}_j = k) = \prod_{K\in\tilde{\mathcal{L}}\backslash\{\{\tilde{K}_j\}\cup Ch(\tilde{K}_j)\}} P(K|Pa(K)) \prod_{K\in Ch(\tilde{K}_j)} P\Big(K|Pa(K)\backslash\{K_j\}, \tilde{K}_j = k\Big), \quad (2.17)$$

where $\tilde{K}_j = k$ translates the causal operation of setting a value k to the exogenous variable $\tilde{K}_j \in \tilde{\mathcal{L}}$. This operation captures the mental action of intervening in the system, in a way that affects only the distribution of $Ch(\tilde{K}_j)$ (Pearl, 2001, 2009; Heckman and Pinto, 2013).

2.4.2.2 Identification

In what follows we establish the identification of the different causal effects of income on fertility in our model. We start with the total effect of income on fertility as defined in Equation (2.9), before the mediated effects through the cost of childbearing and contraceptive behaviour; and then we derive the direct effect of income as a residual.

Our discussion in the previous section suggests that the move from interventional distributions to conditional distributions relies on testable implications as described by marginal and conditional independencies implied by a given hypothetical model. In what follows, we first present, for each causal effect, some testable implications based on the application of the *d*-separation criteria on the relevant hypothetical graphical model. These marginal and conditional independencies will then be used

to discuss the conditions, under which, the interventional distribution $P(Q|\tilde{Y} = y)$ can be represented as a function of conditional distributions. We assume consistency throughout the analysis, in the sense that if we observed a given variable A attain a value a, then $\tilde{A} = a$ and A are the same (Shpitser, 2013). Our analysis is illustrated in terms of discrete variables, where we measure the change in Q produced by a unit change in Y, say from $Y = 0$ to $Y = 1$; but it extends easily to continuous variables wherein integrations replace summations (see Imai, Keele and Yamamoto, 2010).

It is important at this stage to discuss the nature of possible associations in a potential causal model. Graphically, the capacity of a path to carry associations depends both on the orientation of its arrows and on the conditioning variables. Causal paths are sequences of adjacent arrows, all pointing away from the treatment and toward the outcome; all other paths are considered noncausal. Consequently, not all associations between treatment and outcome in a causal model are causal. Some paths may transmit spurious associations. Assuming the necessary variables are observed, the challenge is then to ensure that all spurious associations between treatment and outcome are stopped, without blocking any causal path between treatment and outcome. It follows that identification of causal effects reduces to identifying all possible associations, which are potential sources of spurious associations in the model, and choosing a conditioning set of variables able to block them.

There are only three elementary structural sources of association from which any graphical causal model can be constructed, and each one of them is a potential source of a particular spurious association leading to estimation bias (see Elwert, 2013). In a typical causal model, the first type of association may result from *causation*, as illustrated by a sequential path. An excessive interception of the causal pathway may lead to *overcontrol bias*. In this case, the intercepting variable would remove any

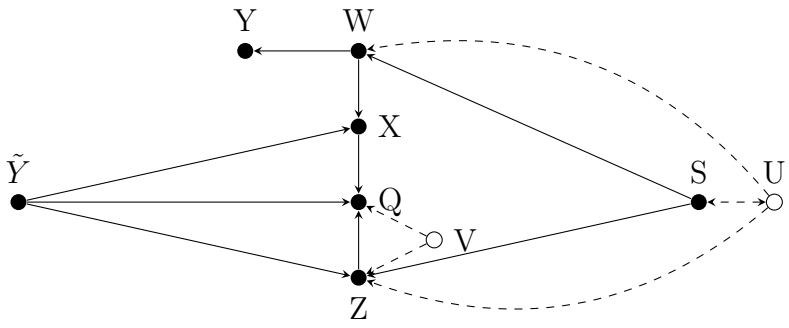

Figure 2.3: Hypothetical Model for the *Total Effect*. Solid bullets represent observed variables, hollow circles represent unobserved (or latent) variables.

causal effect that may exist between two variables. The second source of association is the presence of *confounding* factors, which graphically corresponds to a divergent path. The failure to condition on a common cause creates a *common cause confounding bias*. The third source of association in a causal model is the *common effect*, as described by a convergent path. Conditioning on a common effect, or a descendant of a common effect, produces an *endogenous selection bias*.

Total Effect The following results present marginal and conditional independencies produced by applying the d-separation criteria to the Total Effect hypothetical model in Figure 2.3.

In the hypothetical model for *Total Effect* of Y on Q, (1) $\tilde{Y} \perp\!\!\!\perp W$ and (2) $Q \perp\!\!\!\perp Y|W$.

Proof. In Figure 2.3, each and every path connecting $\tilde{Y}$ to W contains a collider, which suggests that the two variables are marginally independent according to part 1 of the d-separation criteria. The variable Y is connected by a single arrow to the rest of the model through W, so that $Q \perp\!\!\!\perp Y|W$. □

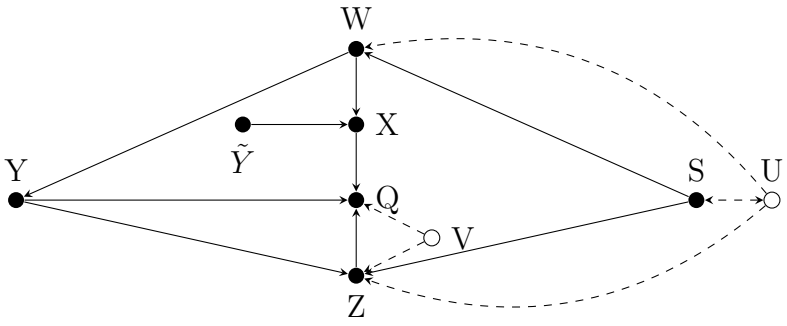

Figure 2.4: Hypothetical Model for the *Price Effect* Fertility Model. Solid bullets represent observed variables, hollow circles represent unobserved (or latent) variables.

Applying these results,

$$\begin{aligned} P(Q|\tilde{Y}=y) &= \sum_W P(Q|\tilde{Y}=y,W)P(W|\tilde{Y}=y) \\ &= \sum_W P(Q|\tilde{Y}=y,Y=y,W)P(W) \\ &= \sum_W P(Q|Y=y,W)P(W). \end{aligned} \tag{2.18}$$

The second equality stems from relationships (1) $\tilde{Y} \perp\!\!\!\perp W$ and (2) $Q \perp\!\!\!\perp Y|W$ of Lemma 2.4.2.2. The third equality comes from applying the consistency assumption. It is easy to show that the *Total Effect* is given by

$$TE = \sum_W \Big[E(Q|Y=1,W) - E(Q|Y=0,W)\Big]P(W). \tag{2.19}$$

Price Effect: PE In the hypothetical model for the *Price Effect* of Y on Q, (1) $\tilde{Y} \perp\!\!\!\perp Q|X,W$, and (2) $X \perp\!\!\!\perp Y|W$.

Proof. In Figure 2.4, $\tilde{Y}$ and Q are d-separated by X,W because X is a collider on the path from $\tilde{Y}$ to Q traversing W. Since Q is a collider, only information on W is needed in order to d-separate X from Y. □

Applying the results from Lemma 2.4.2.2 yields

$$\begin{aligned} P(Q|\tilde{Y}=y) &= \sum_X \sum_W P(Q|\tilde{Y}=y,X,W)P(X|W,\tilde{Y}=y)P(W|\tilde{Y}=y) \\ &= \sum_X \sum_W P(Q|Y,X,W)P(X|\tilde{Y}=y,W)P(W|\tilde{Y}=y) \\ &= \sum_X \sum_W P(Q|Y,X,W)P(X|\tilde{Y}=y,W)P(W) \\ &= \sum_X \sum_W P(Q|Y,X,W)P(X|\tilde{Y}=y,Y=y,W)P(W) \\ &= \sum_X \sum_W P(Q|Y,X,W)P(X|Y=y,W)P(W). \end{aligned} \tag{2.20}$$

In the derivations above, the second equality exploits the fact that conditioning on $\{X,W\}$ d-separates all non-causal paths from Y to Q. In this case, the only source of dependence left between the two is causal. The third equality comes from the fact that W causes Y, which implies that intervening on Y will not influence W. The fourth equality stems from the conditional independence (2) $X \perp\!\!\!\perp Y|W$ of Lemma 2.4.2.2. The fifth equality is as a result of the consistency assumption. Thus, our procedure identifies the *Price Effect* as

$$PE = \sum_X \sum_W E(Q|Y,X,W)P(W)\Big[P(X|Y=1,W) - P(X|Y=0,W)\Big]. \tag{2.21}$$

Contraception Effect: CE In the hypothetical model for the *Contraception Effect* of Y on Q, (1) $\tilde{Y} \perp\!\!\!\perp W$, and (2) $Y \perp\!\!\!\perp Z|W$.

Proof. In Figure 2.5, the path from $\tilde{Y}$ to W contains at least one collider implying that $\tilde{Y} \perp\!\!\!\perp W$. Furthermore, since Q is a collider, only information on W is needed in order to d-separate X from Y. □

Applying results from Lemma 2.4.2.2 yields

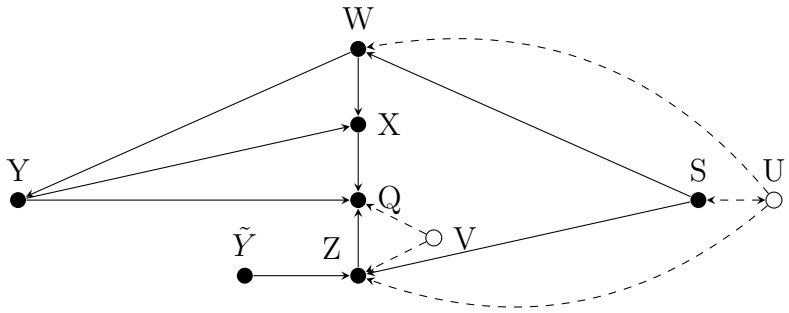

Figure 2.5: Hypothetical Model for the *Contraception Effect*. Solid bullets represent observed variables, hollow circles represent unobserved (or latent) variables.

$$
\begin{aligned}
P(Q|\tilde{Y}=y) &= \sum_Z \sum_W P(Q|Z,W,\tilde{Y}=y)P(Z|W,\tilde{Y}=y)P(W|\tilde{Y}=y) \qquad (2.22)\\
&= \sum_Z \sum_W P(Q|Z,W,Y)P(Z|W,\tilde{Y}=y)P(W|\tilde{Y}=y)\\
&= \sum_Z \sum_W P(Q|Z,W,Y)P(Z|W,\tilde{Y}=y)P(W)\\
&= \sum_Z \sum_W P(Q|Z,W,Y)P(Z|W,\tilde{Y}=y,Y=y)P(W)\\
&= \sum_Z \sum_W P(Q|Z,W,Y)P(Z|W,Y=y)P(W).
\end{aligned}
$$

The second equality comes from applying the *back-door* criterion (Pearl, 1993), which implies that when all spurious paths from Y to Q are disabled – leaving intact all other causal paths between the two – fixing the value of a covariate is the same as conditioning on it. The third equality comes from the relationship (1) $\tilde{Y} \perp\!\!\!\perp W$ of Lemma 2.4.2.2. The fourth equality stems from the relationship $Y \perp\!\!\!\perp Z|W$ of Lemma 2.4.2.2, while the fifth equality results from the consistency assumption. It follows

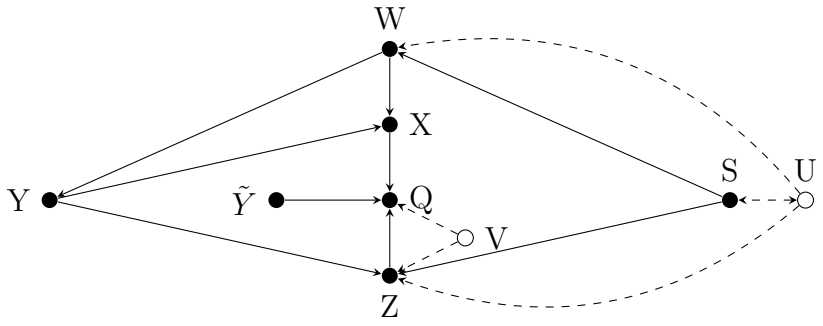

Figure 2.6: Hypothetical Model for the *Direct Effect*. Solid bullets represent observed variables, hollow circles represent unobserved (or latent) variables.

that the *contraception effect* in our fertility model is identified as

$$CE = \sum_{Z}\sum_{W} E(Q|Y,Z,W)P(W)\Big[P(Z|Y=1,W) - P(Z|Y=0,W)\Big], \tag{2.23}$$

which is a function of the observed data.

Direct Income Effect: DIE The identification of the *Direct Income Effect* is relatively more involved compared to the other causal effects identified so far. As illustrated in Figure 2.6, the direct impact of income on fertility is quantified by the level of fertility in a hypothetical situation where we change an individual's level of income, while forcing the cost of childbearing and contraception to behave as if the latter did not change.

In the hypothetical model for the *Direct Effect* of Y on Q, (1) $\tilde{Y} \perp\!\!\!\perp (X,V,Z)$, and (2) $Q \perp\!\!\!\perp Y|X,V,Z$.

Proof. In Figure 2.6, the flow between Y and Q is blocked by the vector of mediator variables $\{X,Z\}$, and the unobserved fecundity V. Moreover, the variables X,W,Z and V are marginally independent from $\tilde{Y}$ because the variable Q connecting $\tilde{Y}$ to the rest of the model is a collider in all four directions. □

Using the results in Lemma 2.4.2.2 yields,

$$\begin{aligned} P(Q|\tilde{Y}=y) &= \sum_{X,Z,V} P(Q|\tilde{Y}=y,X,Z,V)P(X,Z,V|\tilde{Y}=y) \\ &= \sum_{X,Z,V} P(Q|\tilde{Y}=y,X,Z,V)P(X,Z,V) \\ &= \sum_{X,Z,V} P(Q|\tilde{Y}=y,Y=y,X,Z,V)P(X,Z,V) \\ &= \sum_{X,Z,V} P(Q|Y=y,X,Z,V)P(X,Z,V) \\ &= \sum_{X,Z,V} P(Q|Y=y,X,Z,V)P(X|Y,W)P(Z|Y,S,U,V)P(V). \end{aligned} \tag{2.24}$$

The second equality comes from the independence of fertility and income conditional on wage rates, fecundity and contraception as illustrated in the relationship (1) $\tilde{Y} \perp\!\!\!\perp X,V,Z$ of Lemma 2.4.2.2, while the third equality comes from the relationship (2) $Q \perp\!\!\!\perp Y|X,V,Z$ of the same lemma. The fourth equation comes from the assumption of consistency. The fifth equality results from the application of the Markov Factorization property as defined in Equation (2.15).

Note that the last equality in Equation (2.24) cannot be identified using only observed data, since it contains unobservables such as skills, U, and fecundity, V. However, one can still identify the IE as a residual using the following pseudo-additive relation, which captures the decomposition of the total effect into price effect, contraception effect and direct income effect:

$$IE = TE + PE_r + CE_r, \tag{2.25}$$

where PE_r and CE_r are the PE and CE for the reversed transition, from $Y=1$ to $Y=0$ (see Pearl, 2012*b*). Thus, manipulating expressions in Equations (2.23) and

(2.21), and combining them with (2.19) yields:

$$\begin{aligned} IE = \sum_{W} \Big[E(Q|Y=1,W) - E(Q|Y=0,W)\Big]P(W) \\ + \sum_{X}\sum_{W} E(Q|Y,X,W)P(W)\Big[P(X|Y=0,W) - P(X|Y=1,W)\Big] \\ + \sum_{Z}\sum_{W} E(Q|Y,Z,W)P(W)\Big[P(Z|Y=0,W) - P(Z|Y=1,W)\Big]. \quad (2.26) \end{aligned}$$

It is also possible, in the particular case of continuous treatment, to identify directly the direct income effect using control variable methods, such as those discussed in Florens et al. (2008) and Imbens and Newey (2009). Assume, for simplicity, that a woman's heterogeneous skills, U, influence the efficiency of her contraception behaviour, Z, only through the level of education S. Then, under a choice function monotonic in V, the direct income effect could be identified as

$$\begin{aligned} IE = \int\int\int \Big[E(Q|Y=y',X,Z,V) - \\ E(Q|Y=y,X,Z,V)\Big] dF_V(v) dF_{X|Y,W}(x) dF_{Z|Y,S,V}(z), \quad (2.27) \end{aligned}$$

where $dF_A(\cdot)$ and $dF_{A|B}(\cdot)$ represent the distribution function of a random variable A and the conditional distribution function of A given B.

2.4.3 Estimation

Our approach can be empirically implemented using a multistage nonparametric procedure to estimate the different causal effects identified in the previous section. We are interested in the average increase in fertility Q that the transition from $Y = y$ to $Y = y'$ is expected to produce.

2.4.3.1 Total Effect

We assume that the cost of childbearing, X and the efficiency of the contraception method, Z are allowed to track the change in income, Y. In the first step, we estimate the conditional expectations as

$$E(Q|Y = y', w) = g_{y'}(w) \quad \text{and} \quad E(Q|Y = y, w) = g_y(w) \tag{2.28}$$

for every (y', w) and (y, w) cell. In the second step, we estimate the expected value of $g_{y'}(w)$ and $g_y(w)$, respectively, and take the difference

$$TE_{y,y'}(Q) = E_W\Big[g_{y'}(w)\Big] - E_W\Big[g_y(w)\Big]. \tag{2.29}$$

2.4.3.2 Price Effect

Assume now that we wish to hold Y constant, at $Y = y$, and change X to whatever value it would have attained had Y been set to $Y = y'$. This counterfactual definition of the price effect also calls for a three-stage regression. According to Equation (2.21), the first stage consists of estimating the conditional expectation

$$E(Q|Y, X, W) = g(y, x, w) \tag{2.30}$$

for every (y, x, w) cell. In the second step, we sort the estimated conditional expectation with respect to $\{Y, X\}$ so that $g(y, x, w)$ is regarded as a function $g_{y,x}(w)$ of W. We then estimate the expected value of $g_{y,x}(w)$ as

$$f(y, x) = E_W\Big[g_{y,x}(w)\Big]. \tag{2.31}$$

In the third stage, after fixing y and regarding $f(y,x)$ as a function $f_y(x)$ of X, we estimate the conditional expectation of $f_y(x)$, conditional on $Y = y'$ and $Y = y$, respectively, and take the difference

$$PE_{y,y'} = E_{X|Y,W}\Big[f_y(x)|y',w\Big] - E_{X|Y,W}\Big[f_y(x)|y,w\Big]. \tag{2.32}$$

2.4.3.3 Contraception Effect

Similar to estimating the PE, we first proceed by estimating the following conditional expectation as per Equation (2.23)

$$E(Q|Y,Z,W) = k(y,z,w) \tag{2.33}$$

for every (y,z,w) cell. In the second step, we fix (y,z) so that $k(y,z,w)$ is seen as a function $k_{y,z}(w)$ of W. Subsequently, we estimate the expected value of $k_{y,z}(w)$ as

$$m(y,z) = E_w\Big[k_{y,z}(w)\Big]. \tag{2.34}$$

The third step consists of sorting $m(y,z)$ with respect to y to yield $m_y(z)$ before estimating the conditional expectation of $m_y(x)$, conditional on $Y = y'$ and $Y = y$, respectively and take the difference

$$CE_{y,y'} = E_{Z|Y,W}\Big[m_y(z)|y',w\Big] - E_{Z|Y,W}\Big[m_y(z)|y,w\Big]. \tag{2.35}$$

2.4.3.4 Direct Income Effect

Now consider the estimated expected change in Q induced by varying income level from y to y' while keeping constant the cost of childrearing, X and contraception effi-

ciency, Z at whatever values they would have obtained before the intervention. Conceptually, based on results in Equations (2.29), (2.32) and (2.35), the direct income effect (DIE) can be derived as a residual using the following formula:

$$DIE = TE_{y,y'} + PE_{y',y} + CE_{y',y}, \tag{2.36}$$

where $PE_{y',y}$ and $CE_{y',y}$ are reverse causal effect from y to y' for PE and CE, respectively defined as

$$CE_{y',y} = E_{Z|Y,W}\Big[m_{y'}(z)|y,w\Big] - E_{Z|Y,W}\Big[m_{y'}(z)|y',w\Big], \tag{2.37}$$

and

$$PE_{y',y} = E_{X|Y,W}\Big[f_{y'}(x)|y,w\Big] - E_{X|Y,W}\Big[f_{y'}(x)|y',w\Big]. \tag{2.38}$$

2.5 Conclusion

In this chapter, we have introduced an economic fertility model which integrates both the utility maximization and production functions of the household, based on the cost of childrearing and contraception behaviour, subject to unobserved fecundity (see Heckman and Willis, 1976) and unmeasured cognitive and noncognitive abilities (see Heckman, Stixrud and Urzua, 2006).

In particular, our analysis captured the link between labour market and fertility outcomes through family planning and productive time allocation. This is formalized in the concept of a household money income production function (see Schultz, 1961; Ben-Porath, 1967; Mincer and Polachek, 1974), which varies with contraceptive efficiency in line with the human capital theory of demand for health (see Gross-

man, 1972, 2000; Becker, 2007), thus linking access to reproductive health services to participation or performance in the labour market

Using a counterfactual structural estimation framework (see Pearl, 2009; Heckman and Pinto, 2013), we are able to nonparametrically identify and provide steps to nonparametrically estimate the total causal effect of income on fertility, the direct causal effect of income on fertility, and the indirect causal effects mediated through the cost of children and contraception efficiency. We have shown that the sign and magnitude of the total effect of income on fertility depends on the magnitude of the direct effect, which is always positive, and the size of the two indirect effects which are assumed to be negative by definition.

This chapter considers the case of completed family size and did not pay much attention to the dynamic nature of fertility decisions. It is a well known documented fact that the decision to have an extra child, depends on previous parental experience, such that fertility decisions are sequential by nature (Heckman and Willis, 1976; Schmidt, 2008). Moving forward in this thesis, we begin to model some of these dynamic issues.

Chapter 3

The Demand for Reproductive Healthcare

3.1 Introduction

Reproductive health is commonly understood as the capability to procreate, coupled with the freedom to decide if, when and how often to do so. Poor reproductive health outcomes have long been blamed for being one of the main causes of economic hardships among women and their children (see Schultz, 2008). In particular, it has been reported that sexual and reproductive health problems account for 18% of the total global burden of disease, and represent 32% of the burden among women of reproductive age (Singh et al., 2010). In this context, the potential of reproductive healthcare to improve living standards has led to renewed political momentum in the past decade, with policymakers increasingly linking reproductive health issues to development, human rights and gender equity. For example, this momentum delivered changes to the configuration of the Millennium Development Goals, which now include a specific target on universal access to reproductive health care by 2015 (see

Say and Chou, 2011).

Unfortunately, despite the progress made towards making universally available reproductive health care, the achievement so far has fallen well short of expectations (see Fathalla et al., 2006). While the shortfall may be linked to a lack of resources necessary for rolling out reproductive health programs in some countries, the delay experienced in achieving the objective might be symptomatic of how poorly understood are the factors driving the demand for reproductive healthcare. The limited attention the demand for reproductive health services has received in both the theoretical and empirical literatures bears testimony to this fact. The aim of this chapter is to contribute by formalizing the interactions between the various determinants of a woman's reproductive health behaviour during their reproductive years. We focus on family planning services as a specific type of reproductive healthcare.

The current body of knowledge is largely dominated by empirical investigations about the fertility effect of contraception in rich countries (see Bailey, 2006, 2012), and the determinants of unmet need for family planning in poor countries (see Casterline and Sinding, 2000). We argue that these two strands of literature do not explicitly analyze contraceptive behaviour. For example, by their definition, the numbers indicating unmet need for family planning, while useful as a measure of the perceived deficit in the provision of family planning services, they are void of any behavioural content. In particular, knowing the proportion of women who have wanted to stop or delay childbearing but are not contracepting may be an indication of higher costs of access to family planning, but it is not very informative on how the level of contraception efficiency within the very same group of people will be affected, had family planning services been made available at reduced cost. Thus, the main objective of this study is to answer a basic question related to how people adjust their demand for

family planning services, following changes to the their levels of reproductive health capital and some socio-economic outcomes of interest.

The analysis herein describes a theoretical model of the demand for family planning services as a derived demand for reproductive health. In principle, an individual would determine the amount of contraception she will use after balancing the benefits against the costs of a particular service. In particular, the standard consumer theory suggests that increased costs will reduce demand and access to family planning services for poor people. The costs of family planning services include both transaction costs in the form of search and information costs, as well as the monetary and psychic costs associated with using a particular method. Following the tradition started by Grossman (1972, 2000), we consider family planning services as both a consumption and investment good. As a consumer good, contraception is an economic 'bad' – it is potentially a source of discomfort at a particuarly inconvenient time – but it also leads to improvements in reproductive capital stock. As an investment good, the human capital theory suggests that family planning services are an input to the money income production function (Ben-Porath, 1967; Kaestner, 2013) which people maximize subject to the dynamic path of the stock of reproductive health capital.

We begin our analysis by revisiting the canonical Grossman model, but make two observations. First, while healthcare as a final good should enter an individual's utility function, health itself, as one of the multiple forms of human capital, should not be an argument in the individual's utility function, since it can hardly be acquired as a final good (Ben-Porath, 1967; Kaestner, 2013). Thus, in our version of the pure consumption model, healthcare enters the individual's utility function, while health capital appears in the individual money income production function in the pure investment model. We assume that people maximize both models subject to the

dynamic path of the stock of health capital, which depends on changes in both health and healthcare.

The second observation is related to the cyclical and stochastic nature of the human reproduction process. We note that a woman's capability to reproduce and her freedom to plan childbearing largely depend on both biological factors (such as the woman's natural ability to conceive and the characteristics of her partner's semen) and behavioural factors (such as her contraceptive behaviour and sexual practice). It is then natural to postulate that, while being subjected to uncertain shocks stemming from the stochastic nature of the human reproductive process, the stock of reproductive health would improve with experience and knowledge which come with age, in contrast to other forms of health capital (Grossman, 1972, 2000)). In this setting, the role of family planning services, rather than being a direct investment into new reproductive health capital, is mainly to prevent stochastic deterioration of the stock of reproductive health by reducing the probability a woman becomes pregnant.

Consequently, assuming imperfect control over the probability of falling pregnant, our analysis explicitly incorporates into the model the uncertainties surrounding the human reproduction process. The equation of motion in reproductive health is modeled as being partly determined by the deterministic natural investment in reproductive health and partly by a random factor following a standard Brownian motion. The choice of a standard Brownian motion to drive fluctuations in the stock of reproductive health capital is well-suited to the continuous time setting of our model.[1] We then apply stochastic control theory to the pure consumer and investment models, in order to derive analytical solutions that have policy implications.

[1]There are other stochastic extenstions. For example, Laporte and Ferguson (2007) discuss a case where the stochastic nature of health is driven by a random factor with a Poisson distribution, while Liljas (1998) and Picone et al. (1998) introduce uncertainty in the Grossman model through the incidence and size of illness.

The analytical results from this simple model suggest the following theoretical predictions that are testable using real data, one of which is an expected substitution effect, the other follows an intuiton related to goods normality:

- Reproductive healthcare changes inversely to its marginal costs.
- There is a positive relationship between the efficiency of a woman's contraceptive strategy, her natural fecundity rate and frequency of intercourse.

In the empirical part of the study, the theoretical predictions above are tested using individual level survey data from the 2010 Malawi Demographic and Health Survey (2010 MDHS). The theoretical analysis suggests that the relationship between family planning services, their marginal costs and the woman's latent ability to procreate is essentially nonlinear . This raises questions over much of the empirical results from studies using linear functions to estimate the Grossman model. The analysis also suggests that frequency of intercourse might be endogenous to the contracepting decision. Thus we nonparametrically estimate our model of the demand for contraceptive efficiency using the nonseparable empirical framework described in Florens et al. (2008) and Imbens and Newey (2009).

The rest of the work is organized as follows. The next section describes a simple consumer model of healthcare, and derives an expression for the growth rate in the individual demand of medical care. Section 2 reformulates Grossman's pure investment health demand model and uses a path diagram to describe the interactions between medical care and stock of health capital. Section 3 adapts our reformulation of the pure investment model to the demand of reproductive health services by taking into account the stochastic nature of the human reproduction process. Section 4 empirically tests the predictions of our model. Section 5 concludes.

3.2 A Full Health Capital Model

In the original Grossman model, health is treated as a form of human capital from which individuals derive both consumption and production benefits. In this human capital theory of health, the demand for medical care is treated as a derived demand, in the sense that individuals combine medical care with own-time inputs to produce a commodity called *good health*, which in turn yields an output of healthy time. However, in this section, we reformulate Grossman's model by assuming that an individual derives satisfaction from consuming non-medical products and suffers disutility by using healthcare products. Nevertheless, despite being a source of disutility, healthcare underpins the improvement in health status.

3.2.1 Set-Up

Assume that individuals derive satisfaction from two commodities: consumption of non-healthcare products $(C(t))$ and healthcare $(M(t))$, so as to maximize the following life-time utility function

$$\int_0^T e^{-rt} U\Big(C(t), M(t)\Big)dt + e^{-rT} V\Big(K(T)\Big), \tag{3.1}$$

where T denotes total life time, t is measured from the beginning of an employment spell and r is a subjective discount factor with $e^{-rt} > 0$; $V(\cdot)$ is the continuation value. The utility function $U(\cdots)$ exhibits diminishing marginal benefits in $(C(t))$ (see Grossman, 2000), while medical care $(M(t))$ is assumed to be an unpleasant commodity (see Kaestner, 2013) whose additional units decrease satisfaction, $\partial U(\cdots)/\partial M < 0$. It follows that individuals will prefer less medical care to more medical care, implying a negative marginal utility.

At each time t, an individual maximizes the utility in (3.1) subject to the budget constraint

$$Y\Big(K(t)\Big) = p_C C(t) + p_M M(t), \tag{3.2}$$

where p_C and p_M are the respective per unit prices of $C(t)$ and $M(t)$, while income $Y(K(t))$ is assumed to be endogenous and directly dependent on the individual's stock of health capital, $K(t)$.

Furthermore, the stock of good health evolves according to the following equation of motion

$$\dot{K} = H\Big(C(t), M(t)\Big) - \delta(t)K(t), \tag{3.3}$$

where $\dot{K}$ represents the derivative of the health capital with respect to time ($\dot{K} \equiv \partial K/\partial t$), $H(\cdot,\cdot)$ is the individual's current health status, which is assumed to be increasing and concave in healthcare, and $\delta(t)$ the natural rate of health deterioration. Following the installation function approach (see Hayashi, 1982), the transition equation in (3.3) describes changes in health as health capital accumulation net of depreciation. Here, we assume that an individual produces health investment by combining healthcare and other non-medical products.

We also assume that the time available for the different activities is limited. In particular, we posit that Ω, the total time available in any period, is exhausted by the following activities: working $\tau_w(t)$, or being sick or injured $S\Big(K(t)\Big)$. It follows that the time constraint is given by

$$\Omega = \tau_w(t) + S\Big(K(t)\Big). \tag{3.4}$$

Consequently, the budget constraint in (3.2) can be restated as a function of the wage

rate multiplied by the amount time spent not being sick

$$w(t)\big[\Omega - S\big(K(t)\big)\big] = p_C C(t) + p_M M(t). \tag{3.5}$$

For ease of presentation, we combine the constraints in (3.3) and (3.5) by substituting the flow budget for $C(t)$ in the function of gross investment in health. The resulting reformulated optimization problem for a representative agent is given by

$$\int_0^T e^{-rt} U\Big(C(t), M(t)\Big)dt + e^{-rT} V\Big(K(T)\Big), \tag{3.6}$$

subject to the transition equation

$$\dot{K}(t) = G\big(M(t), K(t)\big) - \delta K(t) \tag{3.7}$$

where $G(\cdot,\cdot)$ is the individual's current health status, which is assumed to be increasing and concave in healthcare such that $G_M > 0$, $G_{MM} < 0$, and $\lim_{M\to\infty} G_M = 0$ (see Hayashi, 1982). Thus, the equation for current health status arises by substituting for $C(t)$ in the gross health investment function $H(\cdot,\cdot)$ is

$$G\big(M(t), K(t)\big) = H\left[M(t), \frac{1}{p_C}\Big(w(t)\big(\Omega - S(K(t))\big) - p_M M(t)\Big)\right].$$

For simplicity, we assume that the utility function is homogeneous of degree one, such that the Lagrangian of this maximization problem can be given in intensive form as

$$\mathcal{L} = \int_0^T e^{-rt}\Big[u\Big(m(t)\Big)\Big]dt + \int_0^T e^{-rt}\lambda(t)\Big[g\big(m(t), k(t)\big) - \big(\delta + \gamma\big)k(t) - \dot{k}\Big]dt, \tag{3.8}$$

where all variables are in per unit of consumption of non-medical products and γ is

the growth rate of this consumption. In this setting, $m(t)$ is the control variable and $k(t)$ is the state variable.

In order to solve this problem, we first define the Hamiltonian as

$$\mathcal{H}(m,\lambda,k,t) \equiv u\Big(m(t)\Big) + \lambda(t)\Big[g\Big(m(t),k(t)\Big) - \Big(\delta+\gamma\Big)k(t)\Big]. \tag{3.9}$$

Recasting the Lagrangian in (3.8) in terms of $\mathcal{H}$ gives

$$\mathcal{L} = \int_0^T e^{-rt}\mathcal{H}(\cdots)dt - \int_0^T e^{-rt}\Big(\lambda(t)\dot{k}\Big)dt + e^{-rT}\Big[v\Big(k(T)\Big)\Big], \tag{3.10}$$

where, if we assume that (λ) is differentiable, the second integral can be solved by parts to produce

$$\begin{aligned}\int_0^T e^{-rt}\lambda(t)\dot{k}dt = e^{-rT}\lambda(T)k(T) - \lambda(0)k(0)& \\ - \int_0^T e^{-rt}\dot{\lambda}k(t)dt + r\int_0^T e^{-rt}\lambda(t)k(t)dt.&\end{aligned} \tag{3.11}$$

Stationarity of the Lagrangian requires that the first order partial derivatives be equal to zero. Then, given the initial state k_0, stationarity implies that there exists one function $\lambda(t)$ such that

$$u_m\Big(m(t)\Big) + \lambda(t)g_m\Big(m(t),k(t)\Big) = 0, \tag{3.12}$$

$$\dot{\lambda} - \lambda(t)\Big[r+\delta+\gamma - g_k\Big(m(t),k(t)\Big)\Big] = 0, \tag{3.13}$$

$$\dot{k}(t) - g\Big(m(t),k(t)\Big) + \Big(\delta+\gamma\Big)k(t) = 0, \tag{3.14}$$

$$v'\Big(k(T)\Big) - \lambda(T) = 0, \tag{3.15}$$

where $y_x(\cdot)$ denotes the first derivative of $y(\cdot)$ with respect to x. From (3.12) we have

that

$$\frac{\dot{\lambda}}{\lambda(t)} = \kappa\dot{m} - \beta\dot{k}, \tag{1.12a}$$

where

$$\alpha(m(t)) = \left[\frac{u_{mm}\big(m(t)\big)}{u_m\big(m(t)\big)} - \frac{g_{mm}\big(m(t),k(t)\big)}{g_m\big(m(t),k(t)\big)}\right],$$

is the difference between the relative changes in marginal benefit and marginal discomfort of healthcare, while

$$\beta(m(t)) = \frac{g_{mk}\big(m(t),k(t)\big)}{g_m\big(m(t),k(t)\big)},$$

captures the interaction between healthcare and improvement in an individual's health status. Substituting (1.12a) and (3.45) into (3.13) yields the following relationship for the growth in healthcare

$$\dot{m}(t) = \frac{1}{\kappa(m(t))}\Big[(r+\delta+\gamma) - g_k\big(m(t),k(t)\big) + \beta(m(t))\dot{k}(t)\Big], \tag{3.16}$$

which is a general relationship that should hold for any twice differentiable utility or investment function.

Any model of health seeking behaviour that satisfies the relationship in (3.16) would imply that a person's health seeking behavior is influenced by her own perception of the relative benefit of healthcare. In particular, if individuals dislike the inconvenience associated with healthcare, they will require some benefits to compensate for the discomfort associated with medical care use. In other words, through the factor $\kappa(m(t))$, the result in (3.16) shows that a person's health seeking behavior includes some weighing between the degree of improvement in the health status due to the behavior and the amount of discomfort that one has to endure when consum-

ing medical products. It is this trade-off that ultimately determines the demand for healthcare in response to fluctuations in the stock of health capital.

3.2.2 Application to Reproductive Healthcare

For concreteness, we assume the following constant relative risk aversion (CRRA) utility function in healthcare

$$u\Big(m(t)\Big) = \frac{1 - m(t)^{1-\eta}}{1-\eta}, \tag{3.17}$$

where η is a parameter with any value $\eta > 0$, except for $\eta = 1$, in which case the function takes the form $u(m(t)) = -\log(m(t))$. We thus have that the expressions for the marginal utility and the change in the marginal utility of healthcare are respectively given by

$$u_m\Big(m(t)\Big) = -m(t)^{-\eta} < 0 \quad \text{and} \quad u_{mm}\Big(m(t)\Big) = \eta m(t)^{-(1+\eta)} > 0. \tag{3.18}$$

We further assume that the capital adjustment costs take the following functional form (see Hayashi, 1982; Galama and Kapteyn, 2011)

$$\dot{k}(t) = \varphi m(t)^{\theta} k(t)^{1-\theta} - (\delta + \gamma)k(t), \tag{3.19}$$

where φ is an efficiency factor, θ is the elasticity of health investment with respect to healthcare, $1-\theta$ is the elasticity of health investment with respect to health capital, δ is the natural rate at which health deteriorates.

From (3.19) have the following first-order derivatives

$$g_k = \varphi m(t)^{\theta} k(t)^{-\theta}, \quad \text{and} \quad g_m = \varphi\theta m(t)^{\theta-1} k(t)^{1-\theta}, \tag{3.20}$$

and second-order derivatives

$$g_{mm} = \varphi\theta(\theta-1) m(t)^{\theta-2} k(t)^{-\theta}, \quad \text{and} \quad g_{km} = \varphi\theta(1-\theta) m(t)^{\theta-1} k(t)^{-\theta}, \tag{3.21}$$

such that the value of $\beta(m(t))$ in (3.2.1) is now given by

$$\beta(m(t)) = (1-\theta) k(t)^{-1}. \tag{3.22}$$

In the same fashion, combining some relevant results from (3.18), (3.20) and (3.21) yields the expression below for the coefficient $\alpha(m(t))$ defined in (1.12a)

$$\alpha(m(t)) = (1-\theta-\eta) m(t)^{-1}. \tag{3.23}$$

It follows that the expression for the change in healthcare with respect to time in (3.16) is now reduced to the following homogeneous differential equation

$$\dot{m}(t) = \left(\frac{r + \theta(\delta+\gamma)}{(1-\theta)-\eta} \right) m(t), \tag{3.24}$$

for which the definite solution can be written as

$$m(t) = m(0) \exp\left(\frac{r + \theta(\delta+\gamma)}{(1-\theta)-\eta} t \right), \tag{3.25}$$

where $m(0)$ is the initial value of healthcare.

The result in expression in (3.25) suggets that at any period in time, healthcare use depends on its initial value and it changes, amongst other things, with the rate of natural deterioration of health, δ. However, the specific direction of the change depends on the difference between the elasticity of health investment with respect to health capital, $1-\theta$ and the relative degree of risk aversion, η. If $(1-\theta) > \eta$, then $m(t)$ will be positively related to δ (see Grossman, 1972, 2000). This implies that a person's healthcare use would increase as the deterioration of her health status accelerates. The opposite is true if $(1-\theta) < \eta$.

However, the healthcare model derived in (3.25) may not be suitable to the case of reproductive healthcare, especially regarding the demand for family planning services. The main challenge is that the stock of reproductive health evolves according to a singular equation of motion. Unlike the standard type of health capital, which can be assumed to increase with investments in healthcare and naturally deteriorate at a deterministic rate (see Grossman, 1972, 2000), the ability of a woman to control her own reproductive process naturally grows with factors such as her age, past experiences, and education, while being subjected to random shocks jointly determined by the woman's natural fecundity, sexual behaviour and contraceptive practice.

It follows that the stock of reproductive health capital would be best described as evolving according to a stochastic process.

$$d\log k(t) = g\big(k(t)\big)dt + \big(1-m(t)\big)p(t)x(t)\sigma dB(t), \tag{3.26}$$

where the stock of reproductive health capital is constrained to be positive $k(t) > 0$, $g\big(k(t)\big)$ is natural improvement in reproductive health capital, $0 \leq m(t) \leq 1$ contraceptive efficiency, $0 \leq p(t) \leq 1$ the natural rate of fecundity, $x(t)$ frequency of coital intercourse, σ volatility of the stochastic process, and $B(t)$ is a standard Brownian

motion defined on the probability space $(\Omega, \mathcal{F}_t, P)$, $B = \{\big(B(t)\big), \mathcal{F}_t\}_{t\geq 0}$. We suppose that $\{\mathcal{F}_t\}_{t\geq 0}$ is the augmentation of filtration, which drives the family formation stochastic process.

Note that standard Brownian motion is one of the most common forms of continuous random shocks in the literature, where the relevant variable is subjected to a continuous series of random disturbances and the optimal time path of the control variables must continuously compensate for shocks (see Laporte and Ferguson, 2007). Then the problem to be solved by a given contracepting woman now becomes one of stochastic optimal control, which, by Bellman's principle, should satisfy a nonlinear partial differential equation over the value function, routinely called the Hamilton-Jacobi-Bellman equation and defined as

$$rV(k,t) = \max_{m(t)} \left\{ \frac{1-m(t)^{1-\eta}}{1-\eta} + g(k(t))V_k + \frac{1}{2}\big[\big(1-m(t)\big)p(t)x(t)\big]^2\sigma^2(t)V_{kk} \right\}, \quad (3.27)$$

where $p(t)$ is the woman's natural fecundity, $x(t)$ frequency of intercourse, and the unknown scalar $V(k,t)$ the Bellman *value function*, which represents the income earned in the labour market from starting in state k at time t and controlling the system optimally from then until time T. The partial differential equation in (3.27) gives us the following necessary conditions for optimality

$$\frac{m(t)^{-\eta}}{(1-m(t))} = -p(t)^2x^2(t)\sigma^2V_{kk}. \quad (3.28)$$

Assuming differentiability, it is possible to approximate the value of V_{kk} using *exact* fixed-coefficient Taylor expansions of the general function $V(k(t))$ (see Alghalith, 2009). Let us consider the value function $V(k(t))$. Its second order Taylor expansion

around the average quantity of reproductive health capital is given by

$$V(k(t)) = V(\bar{k}) + V_k(\bar{k})(k(t) - \bar{k}) + \frac{1}{2}V_{kk}(\bar{k})(k(t) - \bar{k})^2 + R(k(t)), \tag{3.29}$$

where $R(k(t))$ is a remainder to be minimized using

$$\min_{k(t)} \left\{ R(k(t)) = V(k(t)) - \left[V(\bar{k}) + V_k(\bar{k})(k(t) - \bar{k}) + \frac{1}{2}V_{kk}(\bar{k})(k(t) - \bar{k})^2 \right] \right\}, \tag{3.30}$$

for which the first order necessary condition yields

$$V_k(k(t)) = [V_k(\bar{k}) - V_{kk}(\bar{k})\bar{k}] + V_{kk}(\bar{k})k(t), \tag{3.31}$$

where the marginal value is positive (*i.e.* $V_k(\cdot) > 0$) and the change in marginal value is negative (*i.e.* $V_{kk}(\cdot) < 0$), because of diminishing marginal returns dictating that the value function be a well-behaved concave function. We also derive the second order necessary and sufficient condition, which allows us to write that $V_{kk}(k(t))$ is a negative constant given by

$$V_{kk}(k(t)) = V_{kk}(\bar{k}) < 0. \tag{3.32}$$

Thus the expression for the optimal contraceptive strategy in (3.28) can be restated as

$$m_C^*(t) = \phi p(t)^2 x^2(t) \tag{3.33}$$

where $m_C^*(t) = m(t)^{-\eta}/(1 - m(t))$ and the constant $\phi > 0$ captures sensitivity of k to changes in the probability of falling pregnant.

The simple model for a woman's contraceptive behaviour derived in (3.33) suggests

the following regarding the demand for family planning services. Firstly, it is clear that natural fecundity is a very important factor in the demand for birth control. As expected, a woman's contraceptive behaviour is influenced by her natural ability to fall pregnant, $p(t)$. This implies that, since female fecundity is related to the menstrual cycle characterized by periods of high fecundity alternating with periods of lower fecundity, contraceptive efficiency will rise when the risk of getting pregnant is higher, and fall during periods of lower perceived risk of pregnancy (or even temporal infertility, due to menstruation or lactational amenorrhea).

Furthermore, the direct relationship between contraception efficiency and natural fecundity is amplified by the frequency of intercourse $x(t)$ and volatility in the stock of reproductive health capital captured by ϕ. The expression in (3.33) suggests that a woman who frequently partakes in sexual activities or who experiences higher volatility in $k(t)$ is likely to use more efficient contraceptive methods.

3.3 Pure Investment Model

In the preceding section, we considered healthcare to be a consumer product, which is a source of discomfort, but also effects the improvement of an individual's health capital stock. However, good health can also be assumed to arise from a pure investment commodity, which determines income or wealth levels. In this context, good health solely determines the total amount of time available for market and nonmarket production activities (see Grossman, 2000).

3.3.1 The Model

In this section, we assume that an individual's money earnings function is given by

$$y(t) = w(t)q\big(k(t)\big), \tag{3.34}$$

where $w(t)$ is the wage rate and $q(k(t))$ is the working time production function, which is a function of a single input *health capital* (k). To allow for multiple periods, suppose that the stock of health capital (k) is long-lived and its evolution is determined by its initial value, $k(0)$, and by the person's demand for healthcare, m. We assume that k is produced with medical care m as a variable input, such that net investment in the stock of health, $\dot{k}$, equals the individual's gross investment in health care minus deterioration in k

$$\dot{k} = g\big(m(t)\big) - n\big(k(t)\big). \tag{3.35}$$

The differential equation in (3.35) describes the change in the stock of health capital k at each point in time, as a function of new investment in health, $g(\cdot)$, and deterioration in the current stock of health capital, $n(\cdot)$. The gross health investment adjustment costs given by the following convex cost function

$$h\big(m(t)\big) \geq 0. \tag{3.36}$$

The gross adjustment costs are sunk costs constrained to be nonnegative, since health capital is attached to an individual and not resellable in the capital market (see Grossman, 1972; Arrow, 2006). It follows that a person's returns to investing in

health at any time t is given by

$$v_t = w(t)q\big(k(t)\big) - h\big(m(t)\big). \tag{3.37}$$

Let R denote the total benefits from following an optimal health investment strategy over a finite horizon $[0, T]$. If t is a *stopping time*(see Stokey, 2009), the individual's total rewards from consuming healthcare $m(t)$ can be thought of as the sum of two terms: expected reward over $[0, t)$ and the *continuation values* over $[t, T)$.

$$R = \text{ returns over } [0, t) + \text{ returns over } [t, T)$$

Now, suppose the person's objective is to maximize the present value of her returns to investment in health. Thus, the individual's problem becomes one of choosing the optimal level of investment in health $g(\cdot)$, in order to maximize R, her total net benefit from avoiding the opportunity costs of ill health, while satisfying her resource constraint. It is assumed that the person discounts future benefits at a subjective constant discount factor r, and the labour market is characterized by perfect competition. Thus, given the information at the beginning of a work interval, $t = 0$, the person's finite horizon dynamic problem is

$$\max_{m(t)} \int_{t=0}^{T} e^{-rt}\left[w(t)q\big(k(t)\big) - h(m(t))\right]dt + e^{-rT}v(k(T))$$

subject to

$$\dot{k} = g\big(m(t)\big) - n\big(k(t)\big)$$

$$m(t) > 0 \quad \text{(control region)}$$

$$k(0) > 0 \quad \text{(given)}$$

where T is the total lifetime and $v(k(T))$ is the *continuation value* or the returns the individual can expect to derive from following an optimal health investment strategy in the future.

In this dynamic problem $m(t)$ is the *control variable*, whereas $k(t)$ is the *state variable*. To derive analytical solutions, we first set up a Lagrangean. For convenience, each constraint is multiplied by the factor e^{-rt} to yield

$$L = \int_{t=0}^{T} e^{-rt}\left[w(t)q\big(k(t)\big) - h(m(t))\right]dt + e^{-rT}v\big(k(T)\big) - \int_{t=0}^{T} e^{-rt}\lambda(t)\big[\dot{k} - g\big(m(t)\big) + n\big(k(t)\big)\big]dt$$

where the *adjoint variable* $\lambda(t)$ is associated with the dynamic constraint in (3.35). It is also called the *co-state variable* and represents the shadow price of k, or the value of a gift of a unit of health capital at time t (Arrow, 2006).

Next the Hamiltonian H is defined as:

$$H(\cdots) = \Big[w(t)q\big(k(t)\big) - h(m(t))\Big] + \lambda(t)\Big[g\big(m(t)\big) - n\big(k(t)\big)\Big]$$

such that the Lagrangian can be re-written as

$$L = \int_{t=0}^{T} e^{-rt} H(m(t), k(t))dt - \int_{t=0}^{T} e^{-rt}\lambda(t)\dot{k}dt + e^{-rT}v(k(T)). \tag{3.38}$$

Suppose that the adjoint variable is differentiable, then it follows that the second component of the right hand side of (3.38) can be integrated by parts to give [2]

$$\int_{t=0}^{T} e^{-rt}\lambda(t)\dot{k}dt = e^{-rT}\lambda(T)k(T) - \lambda(0)k(0) - \int_{t=0}^{T} e^{-rt}k(t)\dot{\lambda}dt + r\int_{t=0}^{T} e^{-rt}k(t)\lambda(t)dt,$$

so that the Lagrangian can be now be restated as

$$L = \int_{t=0}^{T} e^{-rt}\Big[H(\cdots) + k(t)\dot{\lambda} - rk(t)\lambda(t)\Big]dt + e^{-rT}\Big[v\big(k(T)\big) - \lambda(T)k(T)\Big] + \lambda(0)k(0).$$

The *Maximum Principle* requires that, for stationarity, the Hamiltonian be maximized along the optimal path. In our setting, the set of necessary conditions for an optimal solution of the problem in (3.38), which relate to the optimal paths of $m(t)$ and $k(t)$, are given by

[2]Note that

$$\int_{t=0}^{T}\big(\dot{x}z(t) + x(t)\dot{z}\big)dt = \int_{t=0}^{T}\frac{d}{dt}\big(x(t)z(t)\big)dt = x(T)z(T) - x(0)z(0),$$

where the first equality follows from the product rule ($\frac{d}{dt}(xz) = \dot{x}z + x\dot{z}$) and the second from the Fundamental Theorem of Calculus.

$$(i) \quad L_m = H_m(\cdots) = 0$$

$$(ii) \quad L_k = H_k(\cdots) + \dot{\lambda} - r\lambda(t) = 0$$

$$(iii) \quad L_\lambda = \dot{k} - H_\lambda(\cdots) = 0$$

$$(iv) \quad L_{k(T)} = v_k(k(T)) - \lambda(T) = 0$$

Consider the necessary conditions in $(i) - (iv)$. After replacing for the value of the expression of $H(\cdots)$, these conditions imply

$$\lambda(t) = \frac{h_m\big(m(t)\big)}{g_m(m(t))} \tag{3.40a}$$

$$\dot{\lambda} = -w(t)q_k\big(k(t)\big) + \big[n_k(k(t)) + r\big]\lambda(t) \tag{3.40b}$$

$$\dot{k} = g\big(m(t)\big) - n(k(t)) \tag{3.40c}$$

$$\lambda(T) = v_k(k(T)) \tag{3.40d}$$

where $\lambda(t)$ is the shadow price of health capital, $h_m\big(m(t)\big)$ is the marginal adjustment cost, $g_m(m(t))$ is the marginal health benefit of healthcare, $q_k\big(k(t)\big)$ is the marginal production benefit of health capital, and $n_k(k(t))$ is the rate of natural deterioration of health capital.

From (3.40a) we derive the following expression of the growth rate for λ, the shadow price of health capital

$$\frac{\dot{\lambda}}{\lambda(t)} = \left[\frac{h_{mm}(m(t))}{h_m(m(t))} - \frac{g_{mm}(m(t))}{g_m(m(t))}\right]\dot{m}, \tag{3.41}$$

which, when substituted in (3.40b), yields the following equation of motion for health-

care

$$\dot{m} = \frac{1}{\mu}\left[\Big(r + n_k(k(t))\Big) - w(t)q_k(k(t))\frac{g_m(m(t))}{h_m(m(t))}\right], \tag{3.42}$$

where $w(t)q_k(k(t))$ is the marginal revenue benefit of health capital. It represents the monetary value of the marginal production benefit of health capital, while $g_m\big(m(t)\big)/h_m\big(m(t)\big)$ is the shadow price of healthcare indicating the health benefit derived from the last unit of money spent on healthcare. And, the difference between the relative changes in the marginal adjustment costs and marginal health benefit of healthcare is captured by the coefficient

$$\mu = h_{mm}\big(m(t)\big)/h_m\big(m(t)\big) - g_{mm}\big(m(t)\big)/g_m\big(m(t)\big) > 0$$

, since $h_{mm}(\cdot) > 0$ and $g_{mm}(\cdot) < 0$.

The health investment behaviour model described in (3.42) is consistent with the predictions derived from the consumer health seeking behaviour model in (3.16) of Section 3.2. In particular, similar to the consumer model, the expression in (3.42) suggests that growth in healthcare use is positively related to the rate of natural health stock deterioration. Moreover, our health investment behavior model also indicates that an increase in the marginal revenue benefit of health would reduce growth in healthcare use. This implies that, as healthier people earn more, they reduce their level of medical care use, which is consistent with one of the main predictions of the consumer healthcare model we presented earlier, and much of the empirical literature (see Wagstaff, 1986; Liljas, 1998; Zweifel, 2012).

3.3.1.1 Phase Space Analysis

The following analysis uses a phase diagram (see Shone, 2002; Romer, 2006), to discuss the dynamics of a person's health investment strategy. In particular, the expressions in (3.40c) and (3.42) provide a convenient way to describe the demand for medical services in terms of the evolution of m and k. In what follows we consider the solution to our health seeking behaviour as a pair of functions $m(t)$ and $k(t)$, that can be represented by a path which defines a locus in (m, k) space, with the steady state at their intersection.

If we assume, for simplicity, that gross health investment is given by an identity function of the form $g(m(t)) = m(t)$, and that health capital deteriorates at a constant rate δ, so that $n(k(t)) = \delta k(t)$ (see Grossman, 1972, 2000), then the first-order conditions stated in Equation (3.40) simplify to

$$\lambda(t) = h_m\big(m(t)\big) \tag{3.43a}$$

$$\dot{\lambda} = -w(t)q_k\big(k(t)\big) + (\delta + r)\lambda(t) \tag{3.43b}$$

$$\dot{k} = m(t) - \delta k(t) \tag{3.43c}$$

$$\lambda(T) = v_k(k(T)) \tag{3.43d}$$

Condition (3.43a) implies that the shadow price of health capital evolves over time according to the following expression

$$\dot{\lambda} = h_{mm}\big(m(t)\big)\dot{m},$$

which, when substituted into (3.43b), yields

$$\dot{m} = \frac{(\delta + r)h_m(m(t)) - w(t)q_k(k(t))}{h_{mm}(m(t))}.$$

It follows that the dynamics of the optimal health investment strategy would be characterized by the pair of differential equations below

$$\dot{m} = \frac{(\delta + r)h_m(m(t)) - w(t)q_k(k(t))}{h_{mm}(m(t))}, \tag{3.44}$$

$$\dot{k} = m(t) - \delta k(t). \tag{3.45}$$

which divide the space into four regions; in each of which, the paths of m and k have different directions.

The arrows in Figure 3.1 show the directions of motion of both m and k. The steady-state is characterized by both $\dot{m} = 0$ and $\dot{k} = 0$, wherein the stock of health capital deemed optimal, k^*, equates the cost of investment to the marginal returns

$$(\delta + r)h_m(m(t)) = w(t)q_k(k(t)), \tag{3.46}$$

and there is no net investment

$$m(t) = \delta k(t). \tag{3.47}$$

Various trajectories satisfy equations (3.44) and (3.45). However, for any positive initial value of the health capital stock, k_0, there is a unique optimal path leading to the steady-state, while all other paths lead away from the equilibrium. Figure 3.1 describes how m and k must evolve over time to satisfy an individual's intertemporal optimization condition (see Equation 3.44) and the dynamics of her stock of health capital, k (see Equation 3.45).

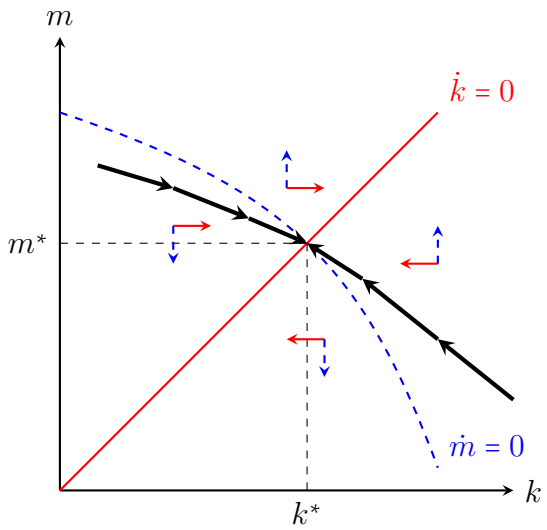

Figure 3.1: Optimal Health Investment Strategy.

The change in healthcare over time, $\dot{m}$, is zero when the marginal cost of medical services is equal to the marginal benefit of further investment in health care. When k exceeds the level that yields $\dot{m} = 0$, m is rising; when k is less than this level, m is falling. For $\dot{k}$ to be zero, investment in m must equal depreciation in k. Thus $\dot{k}$ is zero when $m(t)$ equals $\delta k(t)$. When m exceeds m^*, $\dot{k}$ is positive; when m is less than m^*, $\dot{k}$ is negative.

However, for any positive initial level of k, there is a unique initial value of m which allows the person to move along the *saddle path* to equilibrium. This unique initial value of m is consistent with the person's intertemporal optimization condition and the dynamics of her nonnegative stock of health capital k. Note that, to the right of the $\dot{k} = 0$ line and below the $\dot{m} = 0$ curve, $\dot{m}$ is negative and $\dot{k}$ is negative, suggesting that both m and k are falling, and so the arrows point down and to the left.

3.3.1.2 The Impact of Health Care Costs and Wage Movements

It is also possible to illustrate a number of healthcare issues under deterministic conditions using the phase diagram in Figure 3.1. For example, the following analysis addresses the implications of changes in the cost of healthcare and movements in the wage rate for a person's health investment strategy. Note that the evolution of k, as described in (3.45), depends neither on the cost of health care $h(m(t))$ nor on the wage rate w. Thus, a change in $h(m(t))$ or $w(t)$ will only affect the $\dot{m} = 0$ locus. Recall that the values of m and k where $\dot{m}$ equals zero is defined by

$$(\delta + r)h_m(m(t)) = w(t)q_k(k(t)) \tag{3.48}$$

In this context, an increase in $w(t)$ will shift the $\dot{m} = 0$ locus upward at every level of k. At this point, the assumed convexity of the cost function $h(\cdot)$ implies that its second derivative $h_{mm}(\cdot)$ is positive, while the second derivative of the production function $q_{kk}(\cdot)$ is negative, because of the law of diminishing marginal returns. It follows that, in order to restore the equilibrium in steady-state after an increase in w, m^* has to rise at all levels of k^*, shifting upwards the $\dot{m} = 0$ curve as illustrated in Figure 3.2. Thus, in terms of the phase diagram, a permanent increase in the wage rate leads to a jump in the use of healthcare to a point on the new saddle path (Point E'. Subsequently, k would rise and m moves down along the new saddle path to the new equilibrium (Point E''). Thus the permanent increase in the wage rate moves a person to a new permanent equilibrium, characterized by a high level of medical care and improved health.

In the same fashion, it is straightforward to show that the steady state condition in (3.48) also suggests that an increase in the marginal cost of medical services,

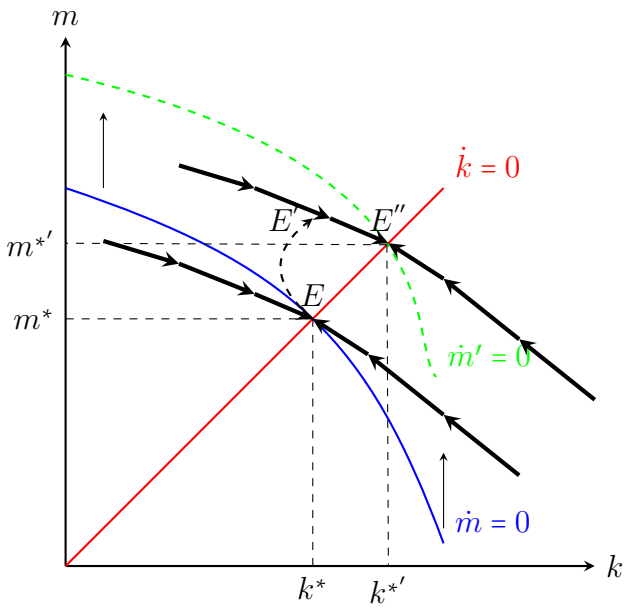

Figure 3.2: The Effect of a Rise in Wage Rate on Health Investment.

$h_m(\cdot)$, will require an increase in the marginal benefit of further investment in health care, in order to restore the equilibrium. This implies a decline in k^*, which causes the marginal benefit to rise, because of the diminishing marginal returns. Thus, a permanent decrease in the cost of medical care will produce the same results as those described in Figure 3.2. In other words, less expensive medical services will encourage investment in health capital, while increasing the cost of healthcare will have the opposite effect, resulting in lower levels of health capital.

3.3.2 Investment in Reproductive Health

This section applies the pure health investment theoretical model developed in Section 3.3 to the analysis of the demand for reproductive healthcare. The theoretical framework allows one to investigate the interactions between labour and reproductive health outcomes, assuming that childbearing and childrearing are time intensive activities. Thus, everything being equal, every fecundable and economically

active woman would have an incentive to take control of her reproductive life by using reproductive healthcare, such as family planning services.

Good reproductive health represents the capability to reproduce and the freedom to decide if, when and how often to given birth. In the case of a fecundable woman, this capability improves over time and is influenced by the random nature of the human reproduction process (see Perrin and Sheps, 1964), characterized by fluctuations in the woman's natural fecundity. We also suppose that these fluctuations can be controlled through a specific contraceptive behaviour. The fact that the woman's rate of natural fecundity is explicitly incorporated into the model, not only accounts for heterogeneity among contracepting women, but also introduces uncertainty into the analysis. Thus, in contrast to the analysis in section 3.3, we allow for a stochastic evolution of reproduction health capital, as a result of uncertainty in the woman's natural fecundity (Heckman and Willis, 1976).

In the rest of the analysis, we focus on reversible contraception as a specific type of reproductive healthcare. Consider a dynamic fertility model in which fluctuations in the reproductive health capital depend on the woman's probability of getting pregnant; and are driven by a standard Brownian motion $B = \{\big(B(t)\big), \mathcal{F}_t\}_{t\geq 0}$ defined on the probability space $(\Omega, \mathcal{F}_t, P)$. The standard Brownian motion is one of the most common forms of continuous random shocks in the literature where the relevant variable is subjected to a continuous series of random disturbances and the optimal time path of the control variables must continuously compensate for shocks (see Laporte and Ferguson, 2007). We suppose that $\{\mathcal{F}_t\}_{t\geq 0}$ is the augmentation of filtration, which drives the family formation process.

Assume a fecundable woman has some control over the fluctuations in the stock of reproductive health capital through her contraceptive behaviour, such that k evolves

according to the following stochastic process

$$d\log k(t) = g\big(k(t)\big)dt + \big(1 - m(t)\big)p(t)x(t)\sigma dB(t), \tag{3.49}$$

where, recall, $g(k(t))$ is the natural improvement in the stock of reproductive health, $0 \leq m(t) \leq 1$ the efficiency of the woman's contraceptive strategy, $0 \leq p(t) \leq 1$ her natural fecundity rate, and $x(t)$ the frequency of sexual intercourse. Note that, both perfect contraception (*i.e.* $m(t) = 1$) or infertility (*i.e.* $p(t) = 0$) would solve uncertainty in the dynamics of $k(t)$.

In this setup, a contracepting woman's optimization problem becomes

$$\begin{gathered} V(t) = \max_{m(t)} \mathrm{E}_0\left[\int_0^t e^{-rt}\big[w(t)q\big(k(t)\big) - h\big(m(t)\big)\big]dt + e^{-rt}v\big(k(t)\big)\right] \\ \text{subject to} \\ d\log k(t) = g\big(k(t)\big)dt + \big(1 - m(t)\big)p(t)x(t)\sigma dB(t) \\ m(t), k(0) > 0 \end{gathered} \tag{3.50}$$

where $h(m(t))$ represents the economic and non-economic costs of contraception to individuals. While economic costs would mainly include monetary expenses, there are significant non-economic costs for using birth control such as the fear of side effects, social disapproval and spousal resistance, as well as unnecessary medical barriers (see Casterline and Sinding, 2000; Bongaarts, 2010).

If we assume that the woman's objective is to maximize the expected current flow of money earnings plus the expected change of future money earnings due to the drifts and volatility of her reproductive health capital (see Stokey, 2009; Bjork,

2009), then the value function V together with the optimal choice of $m(t)$ satisfy the Hamilton-Jacobi-Bellman (HJB) equation

$$rV = \max_{m(t)}\Big\{w(t)q\big(k(t)\big) - h\big(m(t)\big) + \big[g(k(t))\big]V_k + \frac{1}{2}\big[\big(1-m(t)\big)p(t)x(t)\big]^2\sigma^2 V_{kk}\Big\}, \quad (3.51)$$

where $V_k > 0$ and $V_{kk} < 0$.

The first order necessary condition provides an expression for the optimal contraceptive strategy, which implies that at the optimum point the marginal cost of contraception is equal to the marginal benefit of contraception as measured by the reduction in the magnitude of the random shocks to the stock of reproductive health capital

$$h_m(m(t)) = -(1-m(t))p^2(t)x^2(t)\sigma^2 V_{kk},. \quad (3.52)$$

Assume that the woman's total cost of contraception has constant marginal cost π per unit of contraceptive efficiency such that

$$h(m(t)) = \pi m(t). \quad (3.53)$$

It follows that the optimal level of contraceptive efficiency for a sexually active woman would be derived from (3.52) as

$$m^*(t) = 1 + \frac{\pi}{p^2(t)x^2(t)\sigma^2 V_{kk}}. \quad (3.54)$$

Assuming differentiability, it is again possible in this case to use the approximate value of V_{kk} derived in (3.32) to get a closed-form solution for $m^*(t)$. In particular,

plugging in (3.54) results in the following closed form solution for the demand of contraceptive efficiency

$$m_I^*(t) = 1 - \frac{\pi}{\phi p^2(t) x^2(t)}, \tag{3.55}$$

where $\phi > 0$ is a constant.

The model in (3.55), derived using the pure investment model, suggests a couple of interesting predictions which are consistent with the results derived when we applied the full health capital model to the case of reproductive healthcare in section 3.2. Other things being equal, the expression in (3.55) suggests that the efficiency of a woman's contraceptive strategy, $m(t)$, increases with natural fecundity, $p(t)$, and frequency of intercourse $x(t)$. Since natural fecundity is a cyclical event, it is possible that contracepting women will use more efficient contraceptive methods only during the time when they are more at risk of falling pregnant in the menstrual cycle. In this setting, women who frequently have intercourse are also more likely to use relatively more efficient contraceptive methods.

Furthermore, the pure investment model has a clear advantage over a pure consumer model of birth control, in that it explicitly takes into account the impact of the cost of family planning services on a woman's contraceptive behaviour. In particular, equation (3.55) suggests that the efficiency of a woman's contraceptive strategy, $m(t)$, is negatively linked to the opportunity cost of contraception services, π. Faced with higher costs of family planning services, contracepting women will settle for less efficient contraceptive methods. The opposite is also true if family planning services are made more affordable. In the extreme case that the cost of family planning is zero, the model in (3.55) predicts that contracepting women will choose the most efficient contraceptive method available, $m(t) = 1$.

3.4 Empirical Analysis

The economic analysis of a woman's contraceptive behaviour conducted in this study yields expressions of the demand for contraception efficiency, $m(t)$, as nonlinear functions of the observed cost of contraception, $\pi(t)$, frequency of sexual intercourse, $\chi(t)$, and the latent woman's natural fecundity, $p(t)$, as described in (3.33) and (3.55), respectively. The main interest in the following empirical analysis is to test the two econometric models of the demand for family planning services derived from these two expressions using household survey data and compare results to their theoretical predictions.

The nonseparability relationship between the variables in the models (3.33) and (3.55) implies that the effect of a random variation in one factor on contraception efficiency will vary with all the other covariates in the model (see Chesher, 2003). In this context, nonseparability provides for the possibility that otherwise comparable women in terms of one observable will adopt contraceptive methods with different levels of efficiency due to different interactions among the other variables in the model.

The frequency of intercourse captures the essence of a woman's sexual activity (see Brown, 2000). We assume that the choice of the frequency at which a woman has intimate intercourse is endogenous, in the sense that it may be taken jointly with the contracepting decision. We postulate that, while it is true that sexual activity is a *sine qua non* for human reproduction, sexual intercourse is also a source of pleasure and may even be a source of income (see Moffat, 2005; Smith and Christou, 2009; Hakim, 2010). This implies that, otherwise identical women are likely to have different contraceptive strategies for the same level of intercourse frequency.

We argue that, as an instrument for human reproduction, sexual activity will be driven mainly by the ideal number of children. The higher the ideal family size, the

higher the coital frequency and the lower the efficiency of the woman's contraceptive method. In contrast, we expect that, as a source of pleasure or earnings, coital frequency will be positively correlated with contraceptive efficiency. In this case, sexual activity is likely to be negatively linked to the woman's level of income, because on one side, richer women are able to diversify their sources of entertainment, and on the other side, an increase in income will reduce the need to rely on sexual activities as a source of current or future income.

In what follows we attempt to isolate the heterogeneous impact of the frequency of sexual activities on contraceptive efficiency by estimating an econometric model with essential heterogeneity in outcomes (see Heckman, Urzua and Vytlacil, 2006). Furthermore, since the frequency of intercourse is considered to be a continuous treatment, we adopt the control variable approach, which estimates the demand for contraception efficiency based on a triangular simultaneous equation model. The identification and estimation of this type of model with nonseparable disturbances has been discussed at length in the literature (see Chesher, 2003; Florens et al., 2008; Imbens and Newey, 2009).

3.4.1 Econometric Model

A generalized econometric formulation of the economic models developed above is given by the following non-separable model

$$y = (X, \varepsilon), \tag{3.56}$$

where y is the logit of contraceptive efficiency m (see Berkson, 1944), X is a vector of covariates including the endogenous frequency of sexual intercourse $d \equiv \chi(t)$, and ε is a general disturbance vector representing heterogeneous volatility in the value

function. The model in Equation (3.56) is equivalent to a treatment effects model, since it describes a nonseparable outcome model with a general disturbance.

Our empirical strategy is a multistep procedure, which closely follows the control variable technique discussed in Imbens and Newey (2009). In their analysis, the first step consists of building the control variable from the choice equation, before obtaining, in the second step, the conditional expectation of the outcome, given the endogenous variable and the control variable. Furthermore, Imbens and Newey (2009) show that, given a choice equation that is monotonic in a scalar disturbance, the conditional cumulative distribution function of the endogenous variable, given the instruments, is a control variable.

Therefore, assume that the choice of sexual intercourse frequency is described by the following nonseparable treatment choice model

$$d = \varphi(Z, \epsilon), \tag{3.57}$$

where φ is a strictly monotonic function in Z and the scalar unobservable, ϵ, representing the woman's taste for intercourse. The instrument, Z, captures the observed socio-economic environment, which determines a woman's sexual behaviour, but not her contraceptive behaviour, while ϵ captures the latent taste for sexual intercourse. The strict monotonicity in ϵ implies that a heightened state of interest in intercourse will induce higher frequency of sexual contacts, and is essential to the derivation of the control variable. Imbens and Newey (2009) show that, under independence of (ϵ, ε) and Z, the conditional cumulative distribution function (CDF) of d, given Z, is a control variable

$$v = F_{d|Z}(d, Z) = F_{\epsilon}(\epsilon), \tag{3.58}$$

such that conditioning on the control variable v leads to the identification of structural effects of any change in X from the conditional distribution of Y, given X and v. Note that the CDF in (3.58) can be estimated using standard nonparametric procedures, such as the ones outlined in Li and Racine (2008).

In summary, our empirical strategy is carried out in two steps. The first step estimates the control variable from the choice equation, and in the second stage the estimated control variable is plugged into the outcome equation, as an additional covariate, in order to make any change in X causal. In other words, the second step involves estimating the output equation in (3.56) with the control variable estimates $\widehat{v} = \widehat{F}_{d|Z}(d|Z)$ from the first step in Equation (3.58) as an additional co-variate. In particular, we nonparametrically estimate the following logistic outcome model

$$y = g(X, \widehat{v}) + \varepsilon, \tag{3.59}$$

where $y = \log(m/1 - m)$ and $g(\cdot)$ is an arbitrary function estimated from the data.

3.4.2 Data

The information we use for the empirical analysis comes from two modules of the 2010 Malawi Demographic and Health Survey (MDHS): the module on *Reproductive Behavior and Intentions* and the *Contraception* module. The module on reproductive behavior and intentions records answers to questions about fertility history, current pregnancy status, fertility preferences, and the future childbearing intentions of each woman. The contraception module documents answers about knowledge and use of specific contraceptive methods, source of contraceptive methods, exposure to family planning messages, informed choice, and unmet needs for family planning.

We measure contraceptive efficiency using contraceptive use histories collected in the contraceptive calendar of the 2010 MDHS. A standard DHS contraceptive calendar records, for each woman, information about contraceptive status, births, pregnancies, reasons for discontinuing a method, and marital status by calendar month over at least a sixty month period preceding the survey. This information can be used to analyze the efficacy and continuity of women's contraceptive use (see Ali and Cleland, 2010; Ali et al., 2012). For our analysis, we derive the average efficiency of the woman's contraceptive strategy by first, matching each method with its published efficiency during "typical use" (see Trussell, 2011; World Health Organization, Department of Reproductive Health and Research (WHO/RHR) and John Hopkins Bloomberg School of Public Health/Center for Communication Programs (CCP), Knowledge for Health Project, 2011), and then taking the average over the duration of the reproductive calendar. This operation produces contraception efficiency as a continuous variable taking values between 0 and 1: $0 \leq m \leq 1$.

Using data from the United States of America, Table 3.1 shows some standard contraceptive methods against its level of efficiency during "perfect use", when the method is used correctly and consistently as directed, and during "typical use", which is how effective the method is during actual use. In our computation of the average efficiency of a woman's contraceptive strategy, we assume that, since the category "other folkloric contraceptive methods" as recorded in DHS contains family planning methods with no scientific proof to their efficiency, using them is actually not different from not using any contraceptive method at all.

In what follows we present some statistics describing the structure of our data. We start by analyzing the trend in contraceptive efficiency at the aggregate level. Combining information on the effectiveness of standard methods in their typical use

Table 3.1: Contraceptive Efficiency: Rates of Unintended Pregnancies per 100 Women during the First Year of Use

	Rates of Unintended Pregnancies per 100 Women	
Family Planning Method	**Perfect Use**	**Typical Use**
Total Abstinence	0	0
Implants	0.05	0.05
Male Sterilization	0.10	0.15
Female Sterilization	0.50	0.50
IUD	0.60	0.80
Lactational Amenorrhea Method	0.90	2
Injections	0.05	6
Pill	0.30	9
Diaphragm	6	12
Male Condom	2	18
Female Condom	5	21
Withdrawal	4	22
Periodic Abstinence/Rhythm	5	24
Foam and Jelly	18	28
Other Traditional Methods	85	85
No Contraception	85	85

Source: Adapted from Trussell (2011).

from Table 3.1, with data on contraceptive behaviour from the 2010 MDHS reproductive calendar, suggests that most women in Malawi are increasingly using more efficient contraceptive strategies. The percentages in Figure 3.3 show that the average efficiency of the contraceptive behaviour of a woman of reproductive age in Malawi has increased from 2005 to 2010. In 2005 the average efficiency of contraception in the country stood at 28.16%. This percentage slowly progressed to 34.24% in 2008, before jumping up to 37.64% and 41.64% in 2009 and 2010, respectively.

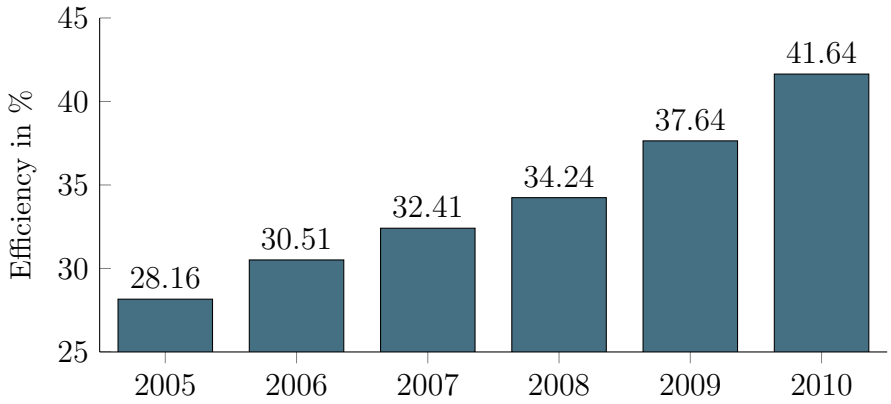

Figure 3.3: Average Contraceptive Efficiency in Malawi 2005-2010

Our theoretical model suggests that these dynamics in contraception efficiency are driven by individual contraceptive behaviours determined by factors, such as fecundity, frequency of intercourse and the cost of contraception. We approximate a woman's natural fecundity by the time-to-first-pregnancy (TTFP), which is defined as the length of time from starting unprotected intercourse till first conception, where larger TTFPs imply less fecund women. The TTFP is mainly determined by biological fecundity, which makes it one of the most common methods for measuring human fecundity (see Basso et al., 2000; Keiding et al., 2012). However, in order to control for the fact that the duration time to first pregnancy might also be influenced by the woman's contraceptive behaviour, we restrict our sample to those married women, whose age at first intercourse coincides with or is higher than the age at the start of the reproductive calendar. The idea is that, since the primary objective of marriage is starting a family, contraceptive use among young married women is likely to be very low before their first pregnancy.

Another variable of interest in our analysis is the frequency of intercourse, which captures a woman sexual behaviour. As a measure of a woman's exposure to sexual activity, coital frequency can be measured by the number of sex episodes during the

woman's last menstruation cycle, or by the length of time since the last episode of sex. The question about the number of sex episodes which appeared in previous DHS has been discontinued, and the current DHS only records the answer to the question probing coital recency. Thus, we use intercourse recency as a proxy for an individual's frequency of intercourse. Furthermore, in our triangular simultaneous equations set-up, coital frequency is an endogenous variable ,which in turn would likely be determined by the ideal number of children, income, as well as the latent taste for sexual intercourse. We use the average years of education between the woman and her partner to approximate the couple's income, since it is not available in the DHS and DHS wealth measures do not necessarily describe current flows.

Finally, another variable of interest in our model is the cost of contraception. Unfortunately, the DHS does not collect information on the cost of family planning services. However, one can infer an index for the combined economic and non-economic costs of contraception in a given geographical area by using the percentage of women with unmet need for contraception (see Casterline and Sinding, 2000; Bongaarts, 2010). The demand for contraception in the DHS is measured by the level of contraceptive use that would prevail if every fecund woman, who wants to avoid pregnancy, were currently using contraception. Couples whose demand for contraception is not satisfied have an 'unmet need' for contraception, and the cost of contraception to individuals is the proximate determinant of the unmet need (see Bongaarts, 2010). Thus, we assume that geographical variations in the percentage of women with unmet need for contraception reveals information about spatial contraceptive cost differentials. In the index, we combine both unmet need for spacing and limiting births. All the covariates used for estimation are summarized in Table 3.2.

Table 3.2: Descriptive Statistics

	Statistics							
	N	**mean**	**min**	**max**	**cv**	**quantiles**		
Variable						p25	p50	p75
Efficiency	1797	0.25	0.15	0.94	0.53	0.15	0.19	0.31
TTFP	1797	17.79	0	100	1.39	0	0	33.33
Recency	1795	12.43	0	31	1.07	1	4	31
% of Unmet Need	1657	11.23	0	55	0.65	6	10	14
Ideal # of Kids	1797	5.76	0	10	0.41	5	6	8
Education	1614	22.95	15.5	44.5	0.13	21	22.5	24.5
Age	1797	3.52	0	7	0.31	3	4	4

Source: 2010 MDHS

3.4.3 Linear Estimation

A preliminary inspection of our data reveals that its structure is consistent with predictions from our theoretical model of contraceptive behaviour. In particular, the correlation coefficients in Table 3.3 suggest that a sexually active woman, who had spent a relatively long period of time before first pregnancy, is more likely to use less efficient contraceptive methods. This is also true for a woman who does not have frequent sexual contacts. Another factor that negatively impacts on the efficiency of a woman's contraceptive behaviour is the level of unmet need for contraception, which is negatively correlated with contraceptive efficiency in our data.

A key finding provided by the correlation matrix is the fact that the ideal number of kids is highly correlated with the frequency of intercourse, but it is not related to contraceptive efficiency. This is very important for our empirical strategy, which relies on an exclusion restriction to control for endogeneity. The positive sign of the

correlation coefficient suggests that a woman, whose ideal number of children is very high, will use less efficient contraceptive methods, and *vice-versa* for a woman who prefers not to have a high number of children.

Table 3.3: Correlation Matrix: These results show only correlation coefficients that are significant at the 5/1% level.

	Efficiency	Unmet	Recency	TTFP	Education	Age	Kids
Efficiency	1						
Unmet	-0.126***	1					
Recency	-0.105***		1				
TTFP	-0.104***	0.049**		1			
Education	0.101***		0.067***		1		
Age	0.194***				0.078***	1	
Kids			-0.073***		-0.118***	-0.054**	1

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

Source: 2010 MDHS

The correlation matrix also shows that education is positively related to both contraceptive behaviour and sexual behaviour in Malawi. The direct relation between education and contraceptive efficiency and intercourse recency is related to the opportunity cost of time: more educated women have a higher opportunity costs of time, which is an incentive to avoid any unplanned exit from the formal labour market, due to accidental pregnancy. The data suggests that they achieve this by adopting more efficient contraceptive methods and reducing intercourse frequency. We also see that the ideal number of children is negatively related to the couple's education, with more educated couples having a lower ideal number of children.

Furthermore, results from a generalized linear model (GLM) estimation (Nelder

and Wedderburn, 1972) of both the pure consumption and pure investment models confirm the strength of each one of these effects as well as its direction. Table 3.4 presents easily interpretable estimates assuming a logit link function with residuals following a binomial distribution. The estimates for the two models with and without unmet need for contraception are very similar. For example, both models predict that, keeping other variables constant, an increase in education by one year will increase the odds of contracepting (with efficiency equal to 1) by roughly 2.7%. We also note from the pure investment model that a one percentage point decrease in unmet need will increase the odds of contracepting (with efficiency 1) by 0.35%.

	(1) Pure Consumption	(2) Pure Investment
Recency	-0.00985***	-0.00932***
	(0.00166)	(0.00165)
TTFP	-0.0132***	-0.0127***
	(0.00282)	(0.00280)
Education	0.0271**	0.0260**
	(0.00976)	(0.00968)
Age	0.0351***	0.0350***
	(0.00739)	(0.00731)
Ideal #Kids	0.00235	0.00491
	(0.0224)	(0.0222)
% Unmet		-0.00356***
		(0.000854)
Constant	-1.816***	-1.767***
	(0.203)	(0.200)
Observations	1487	1487

Standard errors in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 3.4: GLM estimates with logit link function

3.4.4 Nonparametric Estimation

The small correlation coefficients in Table 3.3 suggest that the relationship among the variables could very well be nonlinear, as suggested by the nonseparable expressions in equations (3.33) and (3.55), although it could also point to an endogeneity effect. In this section, we consider nonparametric estimation of a triangular simultaneous equation model of the demand for contraceptive efficiency. In the first step, we use the kernel estimator of Li and Racine (2008) to nonparametrically estimate the conditional cumulative distribution function (CDF) of the logarithm of recency, given the average education of the couple and the woman's ideal number of kids, with the latter used as an instrument.

The results of the kernel regression significance test suggest that the two explanatory variables are highly significant; see Table 3.5. This confirms the highly significant correlation coefficients reproduced in Table 3.3.

Table 3.5: Kernel Regression Significance Test for the Choice Equation.

	Type I Test IID Bootstrap (399 Replications)	
Variable	*Bandwidths*	*p-value*
# of Kids	0.535054	0.0075188**
Education	1.110638	< 2.22e-16***

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Next, for the estimation of the outcome equation, we use local linear estimators as described in Li and Racine (2004), with fixed bandwidths automatically selected through the expected Kullback-Leibler cross-validation method. The results for the significance test in Table 3.6 show that all covariates are highly significant except for the log of recency.

Table 3.6: Kernel Regression Significance Test for the Outcome Equation.

	Type I Test IID Bootstrap (399 Replications)	
Variable	*Bandwidths*	*p-value*
TTFP	3582543	< 2.22e-16***
log(recency)	1959187	0.1553885
% Unmet Need	104.8947	< 2.22e-16***
Age	3.67107	< 2.22e-16***
Education	3.72385	0.0050125 **
vhat	68042.6	< 2.22e-16***

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

We display partial regression plots in Figure 3.4, together with bootstrapped variability bounds, holding all other variables at their respective medians. The plots reveal that that less fecund women (those with longer TTFPs) are likely to use contraceptive methods with lower expected efficiency. Furthermore, contraceptive efficiency decreases with the percentage of unmet need of contraception (i.e. falls with our proxy of cost) and increases with the couple's average level of education. The partial regression plots also suggest that contraceptive efficiency first rises and then falls (maybe) with the average age of the couple, and is non-decreasing with the latent taste for intercourse (as measured by recency of last intercourse).

3.5 Conclusion

Investment in reproductive health has the potential to reduce poverty and avert maternal and childhood deaths, especially in poor countries. In this context, knowledge of factors that affect an individual's reproductive health-related behaviour is

vital in order to understand how an individual values changes in her reproductive health, and creates a basis for normative evaluation of policy interventions aimed at increasing access to family planning and maternal health services. Here we focus on the factors which determine the demand for family planning services, as a particular form of reproductive healthcare.

Very little is known about the factors that determine individual contraceptive behaviour. This is compounded by the fact that the link between medical care and health is a controversial issue in general. The majority of studies on contraception focus more on the fertility effect of contraception and the description of patterns of unmet need for contraception. These largely empirical studies pay little attention to the determinants of contraceptive behaviour, and produce evidence largely dependent on ad hoc models of reproductive behaviour and specific choices on the functional form of estimating equations. Hence, the entire reproductive health agenda appears to lack any credible empirical content.

This study introduces a theoretical reformulation of the original Grossman model, and applies it to the case of reproductive health. We develop two similar models of the demand for contraception efficiency for women of reproductive age using both a pure consumer and investment models for health. In the pure consumer model, individuals maximize a utility function, which has healthcare as input; whereas, in the pure investment model, they are assumed to maximize a money income production function. In both models, the choice of the optimal level of contraceptive efficiency is realized, subject to the equation of motion of the reproductive health capital. The resulting expressions of the demand for family planning services can be used to reproduce some basic stylized facts about individual contraceptive behaviour.

We test the theoretical predictions of our contraceptive behaviour models by fit-

ting individual-level survey data to a triangular simultaneous equations econometric model, with the ideal number of kids as an instrument. The inclusion of the latter is to correct for any possible endogeneity of sexual behaviour. It is assumed that a person's contraceptive behaviour may be influenced by her sexual practice. The theoretical predictions of our model are not rejected by the data. In particular, we confirm that contraceptive efficiency is positively related to the proxy of natural fecundity, but negatively related to the proximate of contraception costs. We also show that contraception efficiency first increases with age before falling, and more educated people tend to use more efficient contraceptive methods.

Access to reproductive health services affords women the opportunity to optimize their lifetime productive opportunities by lowering the costs of long-term career investments and reducing uncertainties regarding the timing of births (see Goldin and Katz, 2002; Canning and Schultz, 2012). Thus, analysing the demand for reproductive health care provides a natural channel through which socio-economic factors can be expected to explain observed fertility outcomes. In the next chapter, we incorporate the model for contraceptive behaviour developed here into an extended framework aimed at analysing the interaction between fertility outcomes, labour market outcomes, and some socio-economic variables of interest for women of reproductive age.

In future empirical work one may be interested in extending our framework to the case of the other components of reproductive health, namely maternal healthcare and child healthcare. It might be possible to also use our framework to analyze the provision of childcare services, as an input to the production function of active time in the formal labour market for working mothers.

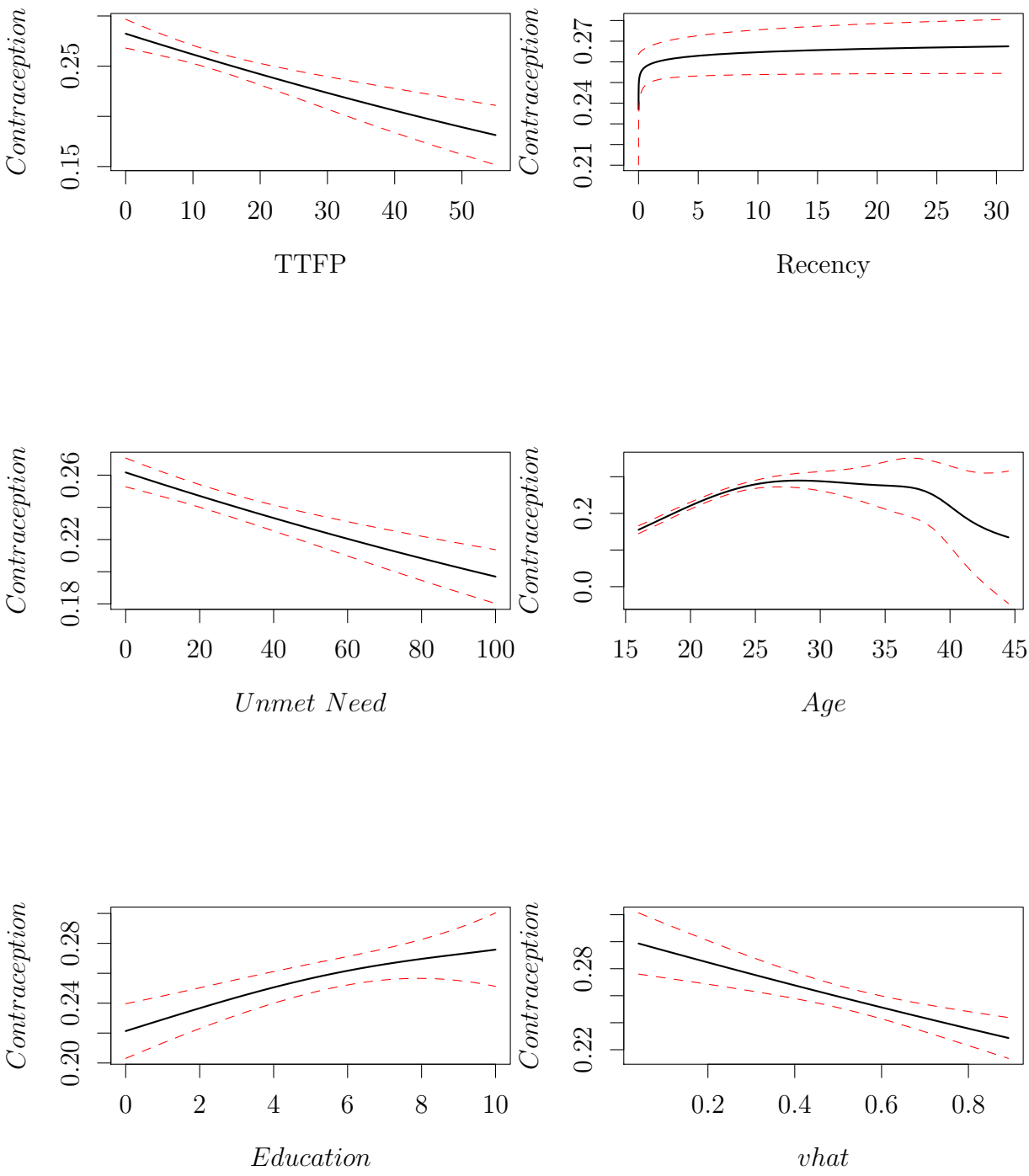

Figure 3.4: Partial Local Linear Nonparametric Regression Plots with Bootstrapped Pointwise Error Bounds

Chapter 4

Family Planning and the Probability of Falling Pregnant

4.1 Introduction

There is a general consensus among population economics scholars that unintended births are problematic. Research has shown that ill-timed childbearing increases fertility and introduces a serious health hazard component into the human reproduction process, while also reducing economic opportunities available to women, preventing their long-term economic prosperity and that of their children (see Gipson et al., 2008). A familiar policy response to the scourge of unintended childbearing is increased access to family planning services (see Canning and Schultz, 2012). However, despite the widespread roll-out of birth control programs, the rates of unintended pregnancy remain stubbornly high, especially in poor communities (see Singh et al., 2010). Thus, the need to evaluate the effectiveness of family planning services in curbing unintended fertility outcomes. In particular, our analysis contributes to the literature on the fertility effect of contraception by discussing the impact of family

planning services on a woman's probability to fall pregnant using information on waiting times to first conception.

Despite the commonly held belief that contraception use gives women control over their fertility outcomes, through the timing of births, the effectiveness of contraceptive use in controlling fertility remains a controversial issue, and is still an under-researched question (see Cleland et al., 2006). It is generally acknowledged that birth control would reduce fertility through two main channels: delay of the onset of childbearing and/or increases in the duration of the interbirth intervals. However, not much attention has been directed towards explicitly understanding the behavioral pathways linking family planning services to birth spacing and timing (see Yeakey et al., 2009).

In addition, theoretical predictions about the impact of contraception on fertility, derived from published economic fertility models, are ambiguous at best (see Bailey, 2012). While earlier analyses intuitively predicted that birth control programs have the potential to decrease childbearing (see Becker, 1960; Michael and Willis, 1976), they often lead to opposite predictions, once they are extended to include some aspects of modern social life, such as uncertainty in the marriage market (Akerlof et al., 1996) and moral hazard dimensions (see Ananat et al., 2009). For example, access to contraception might induce risky sexual behaviour, whereby a contracepting woman would partake in high sexual activity, which might, in turn, cancel out the impact that any increase in the demand for contraceptive efficiency might have, resulting in higher rates of unintended births (Kearney and Levine, 2009). This ambiguity is yet to be resolved conclusively in the empirical literature.

It follows that, without a unified theoretical model to guide empirical inquiries, evaluating the impact of family planning on childbearing has been reduced to an empirical question, which can only be settled by thorough investigation using in-

formation on contraception behaviour and subsequent fertility outcomes. Even so, however, there is very little empirical evidence to support the notion that expanding access to family planning has the potential to reduce rates of unwanted pregnancies (see Bailey, 2012). The scant evidence reported in the literature is largely related to program evaluation, focusing not on behavioural responses to policy changes but mainly on the impact of increased access to family planning services on completed fertility (Kearney and Levine, 2009; Bailey, 2012; Canning and Schultz, 2012).

We argue that, focusing on evaluating the impact of birth control on completed fertility does not take explicitly into account the inherently sequential nature of fertility decisions (see Schmidt, 2008), fails to capture fertility decisions about contraception efficiency and birth timing *as they are being made* (see Iyer and Velu, 2006), and ignores the central role that uncertainty plays in the human reproduction process (see Perrin and Sheps, 1964; Heckman and Willis, 1976). Furthermore, estimates of the effect of family planning on completed fertility, instead of the probability of falling pregnant, is likely to underestimate the impact of birth control on a woman's fertility history. For, even if the availability of family planning services may not change the mind of a woman who has already decided on her optimal completed family size, it gives her at least the control over the timing of each birth in a manner that is more consistent with the history of her economic situation.

When looking for some pattern in any birth history data, one quickly realizes that the number and timing of births is naturally uncertain. One way of dealing with uncertainty in the probability of falling pregnant would be to assume that for every fecundable woman there is an underlying stochastic process leading up to a childbirth. We assume that this latent stochastic process, in reality, describes the dynamics of the agent's reproductive health capital. We define reproductive health

capital as the ability to reproduce and the freedom to plan for childbearing. Our study introduces a simple theoretical framework of sequential fertility behaviour, whose main building block is a stochastic process describing the dynamics of reproductive health capital. Our approach has the advantage of directly relating the timing of fertility decisions to the human capital theory of (reproductive) health (Grossman, 1972). In particular, the analysis takes into account the fact that the duration of a birth interval is significantly influenced by investment and uncertainty in the stock of a woman's reproductive health.

The model describes the way in which a woman's stock of reproductive health evolves over a birth interval, leading up to the possible birth of a child, as a function of her contraception strategy and some random changes in her natural fecundability. Using a mixed hitting time (MHT) framework, we assume that a woman becomes pregnant when the underlying stochastic reproductive health process first crosses a threshold in zero (see Whitmore, 1979; Lee and Whitmore, 2006; Abbring, 2012). In economics, first hitting times naturally arise in structural models, in which agents are assumed to solve an optimal-stopping problem with related rewards described by stochastic processes (see Stokey, 2009). In terms of our model, a woman has some control over the latent stochastic process through her contraception behaviour, which ultimately will have an impact on the average duration of her birth intervals (see Phillips et al., 1988; Pitt et al., 1993; Sinha, 2005). Previous studies that introduced uncertainty in the model for human fertility focus mainly on uncertainty in the characteristics of children (see Ben-Porath and Welch, 1972, 1976; Wolpin, 1984; Leung, 1991; Dahl and Moretti, 2008) and uncertainty in the net benefits of having a child (see Heckman and Willis, 1976; Appelbaum and Katz, 1991; Iyer and Velu, 2006).

This study also extends the current literature on mixed hitting time (MHT) anal-

ysis (see Whitmore, 1979; Lee and Whitmore, 2006; Abbring, 2012) to the case where the distribution of the waiting times associated with the underlying stochastic process is not available in explicit form. In most applications, MHT models serve as an important alternative to proportional hazards models. They provide a modeling structure that is both flexible and realistic enough to incorporate the impact of a variety of observed and unobserved covariates on the statistics of the latent stochastic process (see Lee and Whitmore, 2006, 2010). However, the use of standard MHT models in empirical studies suffers from the limited choice of candidate latent stochastic processes, because the distribution function of waiting times has been given in explicit form only for a very limited number of stochastic processes (Abbring, 2012). Assuming endogenous and heterogeneous treatment effects, we show that waiting times associated with a stochastic process hitting zero can be modeled as a non-separable generalized accelerated failure time model. We then use the control function approach (see Blundell and Powell, 2004; Florens et al., 2008; Imbens and Newey, 2009) in order to identify and estimate the associated hazard structural function (HSF).

In the application section we illustrate the use of our model to analyze the time to the first pregnancy among women from the Democratic Republic of Congo (DRC). This is an attempt to understand the reasons behind the persistently high fertility rates in that country. According to recent research, while most countries have completed or are well advanced in the transition to low fertility, the DRC remains far from meeting the conditions for a sustained fertility transition (see Romaniuk, 2011) with a fertility rate routinely estimated to lie north of 6 children per woman (2007 Democratic Republic of Congo's Demographic and Health Survey).

The rest of the chapter evolves as follows. Section 2 illustrates the sequential nature of the human reproduction process. Section 3 introduces the childbirth hazard

model and presents the derivation of both individual and population childbirth hazard rates. Section 4 discusses the identification and estimation of the fertility effects of family planning. Section 5 illustrates the model with data on time to first birth and covariates from the 2007 Democratic Republic of Congo's Demographic and Health Survey (DRC-DHS 2007). Section 6 concludes.

4.2 The Sequential Nature of Fertility Decisions

The family formation process may be treated as a sequence of reproductive cycles. The inherently sequential nature of a woman's fertility decisions is well-documented in the demographic literature. In particular, it has been suggested that at each decision node, choices about childbearing are made partly in response to experience with previous children (see Ben-Porath and Welch, 1972; Heckman and Willis, 1976; Schmidt, 2008).

In this section we extend the sequential framework to illustrate the impact of the dynamics in a woman's reproductive health capital on the timing of childbearing decisions. The attempt in the present study to apply the MHT framework to the field of population economics is related to the sequential fertility model introduced in Heckman and Willis (1976) and the discrete-time mixture duration model based on a latent process crossing thresholds developed by Heckman and Vytlacil (2007).

We begin by considering the reproductive pattern of a fecundable woman known to be non-pregnant at the onset of sexual activity. At any time after the first intercourse, she can be assumed to be in one and only one of the following reproductive states: she will either be non-pregnant and fecundable (S_0), in the course of a pregnancy (S_1), in the postpartum sterile period associated with non-live birth (S_2) or in the postpartum sterile period associated with live birth (S_3). After spending some random length of

time in either one of the postpartum fertility infecundable states, a woman reverts back to the initial non-pregnant and fecundable state S_0.

It follows that a woman's number and timing of pregnancies and births is completely described by the sequence in which these different reproductive states are visited, and the amount of time spent in each state at each visit. For example, the number of live children ever born to a woman is given by the number of transitions from S_0 to S_3 during her reproductive lifecycle. Similarly, the duration of her first birth interval is equal to the time from first intercourse until the first visit from S_1 to S_3.

To simplify the presentation, we assume that each pregnancy ends in live birth, which allows us to focus on the birth interval given by the duration in the state S_0. We suppose that the length of stay in the reproductive state S_0 is a random variable, whose mean and variance depend on the woman's reproductive health. In particular, the length of time a fecundable woman remains non-pregnant is a random variable, the distribution of which is a function of her use of family planning services.

To fix ideas, we describe a birth interval as a random variable in the simple case of one pregnancy interval as illustrated in Heckman and Willis (1976). The pregnancy interval starts when, at menarche or from a postpartum sterile state, a woman first visits the non-pregnant fecundable state (S_0) in period t. It then passes, after a random period of time δt, into the pregnant state (S_1) at period $t + \delta t$, or at the end of the observation period. Immediately after each non-pregnant fecundable node S_0, the woman is characterized by a certain level of reproductive health capital ($k \geq 0$), which determines her probability of conception. We assume that the woman remains non-pregnant if her stock of reproductive health capital is positive, $k > 0$, but she falls pregnant if this stock is depleted, $k = 0$.

Suppose a fecundable woman in state S_0 is characterized by a natural fecundability $p(t)$ and that she controls her level of reproductive health by investing in family planning services whenever she enters S_0. Under this assumption, the effect of family planning services on the woman's chance of falling pregnant at any time may be expressed as (see Heckman and Willis, 1976)

$$\pi_t = [1 - c(t)]p(t) \qquad \text{for} \qquad c(t) \in (0,1) \quad \text{and} \quad p(t) \in (0,1), \tag{4.1}$$

where $c(t)$ is the contraception efficiency whose value is a function of the technical characteristics of the chosen contraceptive strategy and how well it is used; here $c(t) = 0$ means no contraception and $c(t) = 1$ suggests perfect contraception using a reversible strategy, such as abstinence.

However, as long as the woman remains sexually active, having a nonzero natural fecundability rate means that, despite her choice of contraception, her stock of reproductive health is subject to random shocks both positive and negative. Thus, for a given contracepting woman, the reproductive process can be viewed as an imperfectly controlled stochastic process with an endogenous reproductive transition probability. In this setting, the woman's risk of becoming pregnant increases with the decline in her reproductive health capital, as illustrated in the elementary events tree depicted in Figure 4.1.

4.3 The Childbirth Hazard Rates

In this section, we describe how the hazard rate of a woman falling pregnant varies over the birth interval. We define a birth interval as the length of time between two successive live births, or between a woman's date of first intercourse and the birth of

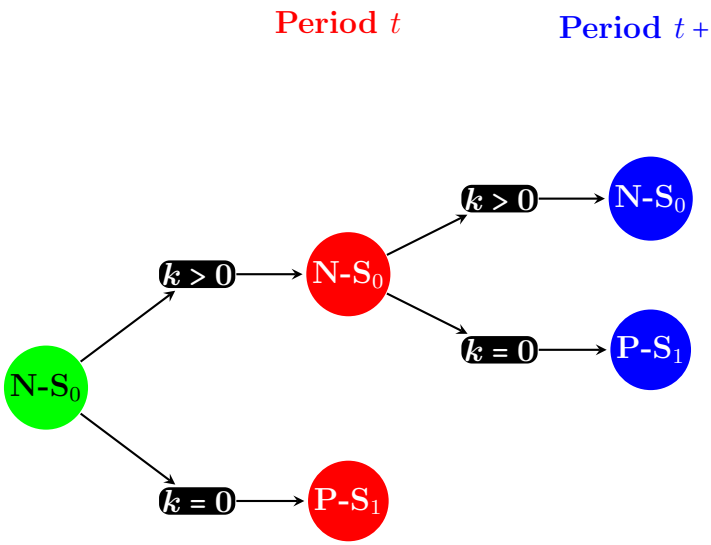

Figure 4.1: Reproductive health capital and pregnancy outcomes within one pregnancy interval.

her first child. Throughout the analysis, the reproductive history data is interpreted as first passage times of a zero threshold, by sample paths of the reproductive health stochastic process in continuous time (see Lancaster, 1972; Whitmore, 1979; Shimer, 2008; Abbring, 2012).

4.3.1 The Model

The analysis makes two key assumptions. *First*, the birth of a child is a probabilistic process and follows a fertility model with two basic components: (a) a parent *latent stochastic process* in time $\{k(t), t \geq 0\}$, given initial value $k(0) = k_0 > 0$, that describes the dynamics of the woman's reproductive health capital; and (b) an *absorbing set* $\mathcal{B}$ in the state space of the unobserved stochastic parent process defining its stopping condition. *Second*, the first hitting time of $k(t)$ to zero is the woman's birth interval duration. It is implied that a sexually active woman would remain in

the non-pregnant stage as long as $k(t)$ stays nonnegative, while the timing of birth coincides with the moment the process $k(t)$ is absorbed in the absorbing boundary of zero.

After the beginning of a birth interval, there exists a latent childbirth risk process represented at each point in time by an index of the woman's stock of reproductive health capital, which satisfies

$$dk(t) = m(c(t))dt - (1 - c(t))p(t)\sigma(t)dB(t), \tag{4.2}$$

where $c(t)$ is the efficiency of the woman's contraception technique; $p(t)$ is her natural fecundity; $\sigma(t)$ is the standard deviation and $B(t)$ is a Wiener process with independent and identically Gaussian distributed (i.i.d) increments $B(t) - B(s) \sim \mathcal{N}(0, t - s)$ for all $t \geq s \geq 0$.

The stochastic process (4.2) for the stock of reproductive health capital specifies the drift $m(c(t))$ and the diffusion coefficient $(1 - c(t))p(t)\sigma(t)$ to be heterogeneous. Abbring (2012) discusses the identification and estimation of a similar model with constant drift and diffusion coefficient that introduces heterogeneity through the threshold or initial condition $k(0)$. Our formulation is consistent with the fact that, in social science settings, at least, a person's behaviour is likely to have a non-negligible impact on the dynamics of the latent stochastic process and, thus, the speed at which the process progresses towards absorption depends on individual heterogeneity.

In our particular case, a given fertility outcome is a function of the woman's contraceptive behaviour that is partly affected by unobserved individual effects, mainly her natural ability to conceive. An increase in $c(t)$ improves the stock of reproductive health capital, while reducing volatility in the stochastic process, which ultimately lengthens the birth interval (and vice-versa for a drop in $c(t)$). However, changes in

the natural fecundity, $p(t)$, act in the opposite direction. An increase in natural fecundity, increases negative shocks to k and makes absorption in zero a certain event in a short period of time. In the extreme case of an infertile woman, with $p(t) = 0$, there are no negative shocks to the process; thus, absorption in zero will never happen.

While the stochastic process $k(t)$ is mostly unobservable for the econometrician, its impact on the reproduction process is measurable through the individual event time $\tau > 0$, when the woman gives birth. We are interested in the probability that the woman gives birth at $t + \Delta t$, given that she is not pregnant in duration τ at t. By assuming $t_0 = 0$, the solution of (4.2) takes the form

$$k(t|k_0, c, p) = k_0 + \int_0^t m(c(s))ds - \int_0^t (1 - c(s))p(s)\sigma(s)dB(s), \tag{4.3}$$

where t is time and k_0 is the random initial stock of reproductive health, and is assumed independent of $B(t) - B(0)$. Using Brownian motion with a drift has the advantage of allowing us to use the well-known analytical result that the first-hitting time of a Brownian motion has an inverse Gaussian probability distribution, whose theoretical properties have been established in the literature (see Lancaster, 1972; Chhikara and Folks, 1989; Molini et al., 2011).

A measure of the relative risk of giving birth is given by the distance between the starting point k_0 and the point of absorption of the latent process $k(t)$. The evolution of the associated conditional probability density function (pdf) can be described by the following Fokker-Plank (FP) equation

$$\frac{\partial p(k, t|k_0, c, p)}{\partial t} = -m(c(t))\frac{\partial p(k, t|k_0, c, p)}{\partial k} + \frac{1}{2}\Big[(1 - c(t))p(t)\sigma(t)\Big]^2 \frac{\partial^2 p(k, t|k_0, c, p)}{\partial k^2}, \tag{4.4}$$

where $p(k, t|k_0, c, p)$ is the transition pdf with initial condition $\delta(k - k_0)$ at t_0.

4.3.2 The Hazard Rates of Giving Birth

The time, τ, at which the stochastic process k starting at $k_0 > 0$ reaches the absorbing barrier $k(\cdot|k_0, c, p) = 0$ for the first time, is also a random variable, such that (4.4) can be solved for $p(k, t|k_0, c, p)$, with the boundary condition $p(0, t|k_0, c, p) = 0$ and the additional condition of $k = +\infty$ being a natural boundary. Under these two boundary conditions, assuming proportionality between the drift and the diffusion coefficient, one can use the method of images to get the general solution for (4.4) as (see Molini et al., 2011)

$$p(k, t|k_0, c, p) = \frac{1}{2\sqrt{\pi A(t)}} \left\{ \exp\left[-\frac{(k - k_0 - B(t))^2}{4A(t)} \right] - \exp(-k_0 q) \exp\left[-\frac{(k + k_0 - B(t))^2}{4A(t)} \right] \right\}, \tag{4.5}$$

where

$$\begin{aligned} A(t) &= \frac{1}{2} \int_0^t (1 - c(s)) p(s) \sigma(s) ds, \\ B(t) &= \int_0^t m(c(s)) ds, \\ B(t) &= q A(t). \end{aligned}$$

The drift $m(c(t))$ quantifies the rate at which the woman approaches the threshold that triggers childbirth. However, there is no guarantee that the process will reach the boundary set $\mathcal{B}$. We recognize the fact that for some women the childbirth risk process $k(t)$ may diffuse away from the childbirth threshold (*i.e* zero) for a long time, while for others it would diffuse almost directly toward zero. The main assumption is that the consistent use of family planning services keeps the stochastic process far from zero for a prolonged period of time, which reduces the probability of falling pregnant and translates into longer birth intervals.

Consider one fecundable woman for a moment. As she progresses through time, her stock of reproductive health randomly fluctuates, due to the combination of two factors: investment in family planning services and random changes in her natural ability to procreate. The woman might experience a relatively steady decline in her ability to control the reproductive process; thus, it eventually hits zero, the level at which we assume she gives birth. This is plausible, for example, when investment in family planning is lower than the deterioration of reproductive health capital. On the other hand, the woman may have experienced a relatively steady increase in her level of reproductive health, in which case, she may never theoretically give birth. This will be the case when the woman perfectly contracepts $c(t) = 1$ or is infertile $p(t) = 0$.

If $m(c(t)) > [(1-c(t))p(t)\sigma(t)]dB(t)$ the risk process will tend to drift away from the threshold, since it implies that some women will choose not to have an extra child by investing in more efficient family planning services. This would also be the case when some women are temporarily infertile, $p(t) = 0$, such that $\tau = \infty$. To use the terminology in Abbring (2012), infertile women make up an unobserved subpopulation that may be described as *stayers*, while those women who might choose not to have a child can be considered *defecting movers*; $\tau = \infty$ with positive probability when $c(t) = 1$. On the other hand, if $m(c(t)) < [(1 - c(t))p(t)\sigma(t)]dB(t)$, the changes to $k(t)$ are negative, and there is a tendency to drift towards the childbirth threshold of zero. In this case, childbirth is almost a certain event that will occur within some finite time with probability one.

In the simple case, where we assume that the speed at which the stochastic reproductive health process evolves and its volatility is constant, the transition probability

is given by

$$p(k,t|k_0,c,p) = \frac{1}{\sqrt{2\pi\Omega^2 t}}\left\{\exp\left[-\frac{(k-k_0-\mu t)^2}{2\Omega^2 t}\right] - \exp\left(-\frac{2k_0\mu}{\Omega^2}\right)\exp\left[-\frac{(k+k_0-\mu t)^2}{2\Omega^2 t}\right]\right\}, \tag{4.6}$$

where $\Omega = (1-c_0)p_0\sigma_0$ and $\mu = m(c_0)$. Then, the conditional survival probability can be derived as

$$S(t|k_0,c,p) = \int_0^{+\infty} p(k,t|k_0,c,p)dk = \Phi\left\{\frac{\mu t + k_0}{\Omega\sqrt{t}}\right\} - \exp\left(-\frac{2k_0\mu}{\Omega^2}\right)\Phi\left\{\frac{\mu t - k_0}{\Omega\sqrt{t}}\right\}, \tag{4.7}$$

where Φ is the standard normal distribution, and the first passage time probability density $f(t|k_0,c,p)$ is the rate of decrease in time of $S(t|k_0,c,p)$

$$f(t|k_0,c,p) = -\frac{\partial}{\partial t}S(t|k_0,c,p) = \frac{k_0}{\Omega\sqrt{2\pi}t^{3/2}}\exp\left[-\frac{(k_0+\mu t)^2}{2\Omega^2 t}\right]. \tag{4.8}$$

It is then straightforward to derive the theory-based conditional hazard rate for the time to childbirth. The conditional birth hazard rate $\lambda(\cdot|k_0,c,p)$ is defined as the instantaneous probability that a birth interval τ will end within the next moment in time:

$$\lambda(t|k_0,c,p) = \lim_{\Delta t\to 0}\frac{\Pr(\tau \le t+\Delta t|\tau > t)}{\Delta t} = \frac{f(t|k_0,c,p)}{S(t|k_0,c,p)}. \tag{4.9}$$

Figure 4.2 illustrates the fact that the hazard rate of childbearing is a function of a woman's initial stock of reproductive health capital as she enters a given birth interval. It also shows that regardless of the initial value of reproductive health capital, all hazards for the Brownian motion converge to the same limiting hazard. At time $t = 0$, if the woman starts with a stock of $k(t)$ close to zero, relative to the quasi-stationary distribution, the childbirth hazard rate is essentially decreasing; whereas, if she starts at a value of k_0 far from zero, the childbirth hazard rate is

essentially increasing (see Aalen et al., 2008).

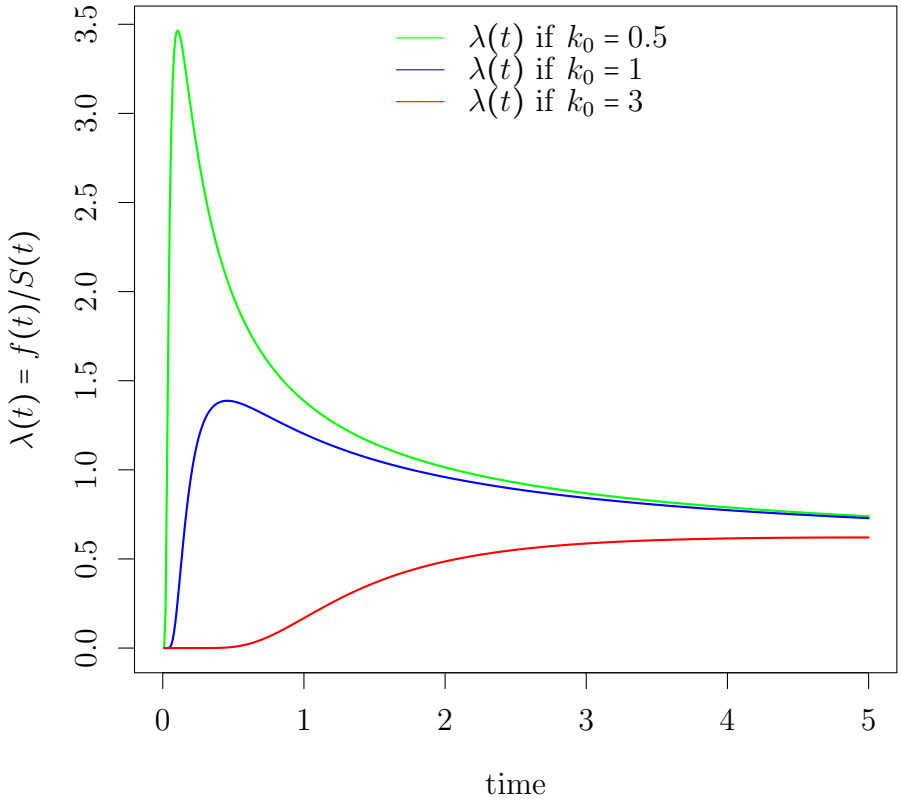

Figure 4.2: Hazard rates for time to childbirth $\lambda(t)$ when the process starts in different values of k_0, assuming $c_0 = 0.5$, $m(c_0) = 1$, $p_0 = 1$ and $\sigma_0 = 2$.

4.4 The Fertility Effect of Family Planning

This section analyzes the fertility impact of family planning services. The goal is to discuss the identification of the causal effect of contraception on the hazard of giving birth by time t using a generalized regression model for birth intervals. We first generalize the theoretical framework presented earlier by relaxing the propor-

tionality condition between the drift and the diffusion coefficient. We then discuss the identification of the fertility impact of family planning in a triangular model with non-separable unobservables (see Chesher, 2003; Florens et al., 2008; Imbens and Newey, 2009).

In section 4.3, the derivation of the statistics of the distribution of the birth interval, τ, is based on the assumption that $A(t)$ and $B(t)$ are proportional (see Molini et al., 2011). This assumption is unconcerned with the qualitative nature of the information about economic processes that economic theory provides. In particular, the rather strong and unpalatable proportionality restriction is unlikely to hold in the case of a fertility model, since it requires a fixed ratio between two functions – one, an unobserved biological process, $p(t)$, and, two, a behavioural factor, $c(t)$.

4.4.1 The Outcome Equation

Suppose that all pregnancies end in live births. Then, the duration of a birth interval is an observation on a random variable, τ, that could be characterized as

$$\tau = \inf_{t \geq 0} \{t : k(t|k_0, c, p) = 0\}. \tag{4.10}$$

Variation in the triplet (k_0, c, p) corresponds to heterogeneity in individual hitting time,s τ. Consequently, a natural way of generalizing the analysis in the previous section is by modeling birth intervals as following a distribution, such that the outcome equation

$$\log(\tau) = \phi(X, \varepsilon), \tag{4.11}$$

is a generalized regression model (see Han, 1987; Ridder, 1990), where ϕ is a twice continuously differentiable birth control production function, which is non-separable

in the unobservable ε. The vector X of covariates includes the woman's endogenous choice of contraception efficiency c. Furthermore, ε is a two dimensional disturbance vector (see Chesher, 2003), which captures the individual unobserved heterogeneity determined by the woman's initial stock of reproductive health capital, k_0, and her natural ability to conceive, p.

In this non-additive formulation, the fertility impact of family planning services on birth intervals would vary with the woman's unobserved natural fecundity and her initial stock of reproductive health. It is also likely that a contracepting woman will choose which family planning services to use, with at least a partial knowledge of her natural fecundability, so that X is correlated with ε.

Our approach to identification and estimation is a multistep process based on control variables (see Blundell and Powell, 2003; Florens et al., 2008; Imbens and Newey, 2009). The control variable is built in the first stage. The second stage derives the conditional distribution of the outcome, given the endogenous variable and the control variable. The hazard function is then constructed. Various contraception fertility effects are recovered by averaging over the control variable or over both the endogenous and control variable.

For the non-separable model, $\tau = \phi(X, \varepsilon)$, we define a control variable as any variable V that, when conditioned upon, produces the independence between X and ε which, in turn, makes changes in X causal, leading to the identification of structural effects. In what follows, the control variable is constructed as a one-to-one function of the unobservable factor in the model for the choice of a contraception technique, which must satisfy the following condition:

Assumption 4.4.1. Control variable*: X and ε are independent, conditional on V (see Imbens and Newey, 2009).*

4.4.2 Choice of a Contraception Method

Assume that the degree of efficiency of family planning services varies continuously on the unit interval $[0, 1]$, with 0 representing ineffective birth control and 1 denoting perfect contraception. This produces a group of *untreated* individuals, whose level of treatment is zero; and a *treatment* group, whose dose of treatment is greater than zero. It is assumed that people in the two groups may react differently to both specific unobservables and the intensity of treatment.

The assumption of a continuum of values for family planning efficiency implies that the latter can be represented as a transformation of some observed covariates and a scalar continuous unobservable term. Then, we denote the assignment mechanism to a certain intensity of treatment as

$$c = h(Z, \eta), \tag{4.12}$$

where c is contraception efficiency, Z are observed covariates, such as schooling status and labour force participation, which may influence the choice of contraception efficiency, but do not have any direct impact on the dynamics of reproductive health capital described in (4.2); η is a continuous scalar variable which measures unobserved subjective benefits from contraception.

Moreover, it is likely that an individual who stands to benefit more from contraception will use more efficient contraceptive techniques. This implies that the intensity of contraception efficiency c increases with the unobservable benefits from contraception η, leading to the following assumption:

Assumption 4.4.2. *η is absolutely continuous with respect to Lebesgue measure;* h *is strictly monotonically increasing in η and* $\mathrm{Z}(\varepsilon, \eta)$ *(Florens et al., 2008).*

Since contraception efficiency is a continuous random variable, it is possible to derive, under the independence of (ε, η) and Z, the control variable, V, as the cumulative distribution function of η. Imbens and Newey (2009) show that the uniformly distributed variable $V = F_{c|Z}(c, Z) = F_{\eta}(\eta)$, where $F_{c|Z}(c, Z)$ is the conditional cumulative distribution function (CDF) of c given Z and $F_{\eta}(\eta)$ is the CDF of η, is such a control variable. Therefore, conditional on the unobserved woman's natural fecundity, η, the demand for contraception efficiency will only depend on exogenous behavioural factors, such as schooling and working status, included in the vector Z.

Theorem 4.4.1. *Suppose the* full independence *condition* $Z(\epsilon, \eta)$ *and strict* monotonicity *of* $h(Z, \eta)$ *with respect to a continuously distributed scalar* η. *Thus, in the triangular model of equations (4.11) and (4.12),* X *and* ε *are* independent, *conditional on* $V = F_{c|Z}(c, Z) = F_{\eta}(\eta)$ *(Imbens and Newey, 2009).*

4.4.3 Identification of the Fertility Effect of Birth Control

The value of a structural feature in a model can be identified by a functional of the conditional distribution function of outcomes, given covariates, under the restriction that this functional returns the same value of the structural feature in all structures admitted by the model (see Chesher, 2003).

Under assumption 4.4.1, it is the case that for any integrable function $\Pi(t)$,

$$\begin{aligned} E[\Pi(\tau)|X = x, V = v] &= \int \Pi\Big(\exp(\phi(x, e))\Big) F_{\varepsilon|X,V}(de|x, v) \\ &= \int \Pi\Big(\exp(\phi(x, e))\Big) F_{\varepsilon|V}(de|v), \end{aligned} \tag{4.13}$$

where x, v, and e are possible values of X, V and ε, respectively; so that changes in x in $E[\Pi(\tau)|X = x, V = v]$ are causal and correspond to changes in x in $\exp[\phi(x, \varepsilon)]$.

This corollary of the strong exclusion restriction in assumption 4.4.1 has been the foundation of various identification results in the presence of a control variable, including the average structural function (Blundell and Powell, 2003) and the quantile structural function, as well as average and policy effects (Imbens and Newey, 2009). Our analysis focuses on hazard effects using the hazard structural function (HSF) $\lambda_\tau(t,x)$, which we define as the hazard rate of $\exp(\phi(x,\varepsilon))$.

The conditional hazard rate $\lambda_{\tau|X}(t|x)$ is defined as the instantaneous probability that a duration τ will end within the next instant of time:

$$\lambda_{\tau|X}(t|x) = \lim_{\Delta t \to 0} \frac{\Pr(\tau \leq t + \Delta t | \tau > t, X = x)}{\Delta t}, \quad [t > 0]. \tag{4.14}$$

For convenience, we discuss the identification of the HSF using the conditional CDF. Note that the conditional hazard is uniquely determined by the conditional CDF, as expressed in the relationship

$$\lambda_{\tau|X}(t|x) = -\frac{\partial}{\partial t}\left[\log(1 - F_{\tau|X}(t|x))\right]. \tag{4.15}$$

Then, for a functional $\Pi(\tau) = \mathbf{1}(\tau \leq t)$, equations (4.11) and (4.13) yield

$$F_{\tau|X,V}(t|x,v) = \int \mathbf{1}\left[\exp\left(\phi(x,e)\right) \leq t\right] F_{\varepsilon|V}(de|v), \tag{4.16}$$

where identification of structural effects requires that the control variable V be integrated out. The integration over the marginal distribution of V requires the following large support condition (Heckman and Vytlacil, 2001; Imbens and Newey, 2009):

Assumption 4.4.3. Common Support*: For all $X \in \mathcal{X}$, the support of the control variable* V*, conditional on* X*, is the same as the marginal support of* V*.*

Then, under assumption 4.4.3, taking the integral of both sides in (4.16), with respect to V, gives

$$\int F_{\tau|X,V}(t|x,v)(dv) = \Pr[\exp(\phi(x,\varepsilon)) \leq t]. \tag{4.17}$$

It follows from the definition of the HSF that

$$\lambda_\tau(t,x) = \frac{\int \left[\frac{\partial}{\partial t} F_{\tau|X,V}(t|x,v)\right](dv)}{\int [1 - F_{\tau|X,V}(t|x,v)](dv)}, \tag{4.18}$$

which is identified over the entire support of the marginal distribution of V, under the common support condition in assumption 4.4.3. Thus, we arrive at the following result:

Theorem 4.4.2. Identification of the HSF*: Consider the latent stochastic process in (4.2) with the associated hitting times described in (4.11). The structural feature* $\lambda_\tau(t,x)$ *is identified for all* $x \in \mathcal{X}$*, under Assumptions 4.4.1 and 4.4.3.*

4.4.3.1 Policy Effects

We now turn our attention to the fertility effects of changes in the choice of birth control methods. For simplicity, suppose contraception efficiency is the only covariate in the outcome equation and the control variable is proportional to unobserved benefits from contraception, so that $x = c$ is a scalar and η is normalized to V.

Consider a policy that seeks to reduce the cost of using family planning services by subsidizing access to condoms in order to achieve total condom coverage among women who are currently not using any modern contraception method. This imposes an efficiency floor $\bar{x}$ on the choice variable X, suggesting a new choice equation of the

form

$$\bar{h}(z,v) = u(h(z,v)). \tag{4.19}$$

If we assume that the support of (X,V) includes the support of $(\bar{h}(z,V),V)$, then it is possible to derive the conditional hazard rate, after a change to $\bar{h}(z,v)$ from equation (4.18) at a given z as

$$\bar{\lambda}_\tau(t,x) = \frac{\int f_{\tau|X,V}(t|\bar{h}(z,v),v)F_V(dv)}{\int S_{t|X,V}(\tau|\bar{h}(z,v),v)F_V(dv)}, \tag{4.20}$$

where $f_{\tau|X,V}(\cdot)$ is the conditional density function and $S_{\tau|X,V}(\cdot)$ is the conditional survival function. A hazard policy effect is then given by

$$\bar{\lambda}_\tau(t,x) - \lambda_\tau(t,x). \tag{4.21}$$

4.4.4 Estimation Strategy

Let τ be a random variable representing survival time to next childbirth. We consider the situation where the birth intervals of interest, τ, are possibly not completely observed, because of censoring. We assume a random-right censoring model with censoring times Ξ. This assumption generates the observed duration $T = \min(\tau,\Xi)$ and the associated censoring indicator $\delta = \mathbf{1}(\tau \leq \Xi)$. Moreover, it is assumed that, given a vector of covariates X and a control variable V, the random variables $\tau|X,V$ and $\Xi|X,V$ are independent. We adopt a multistep approach to the estimation of the HSF from a sample of independent and identically distributed (i.i.d) observed data (T_i,X_i,Z_i,δ_i) $(i = 1\cdots n)$.

The first step estimates the control variable, $\widehat{V}_i$, as the conditional CDF of the scalar endogenous variable, X_i, representing the choice of contraception efficiency

given Z_i (see Imbens and Newey, 2009)

$$\widehat{V}_i = \widehat{F}_{X|Z}(X_i, Z_i). \tag{4.22}$$

These estimates are then used in subsequent steps with the goal to construct estimators of interest related to the derivation of the estimator $\widehat{\lambda}_\tau(t,x)$ of the hazard rate function $\lambda_\tau(t,x)$. In particular, from the conditional independence of τ and C, we have that the estimate of the conditional survival function is given by

$$\widehat{J}_{\tau|X}(t,x) = \int \widehat{S}_{\tau|X,\widehat{V}}(t|x,\widehat{v})(d\widehat{v}) \int \widehat{G}_{\Xi|X,\widehat{V}}(\xi|x,\widehat{v})(d\widehat{v}), \tag{4.23}$$

where $\widehat{S}_{\tau|X,\widehat{V}}(\cdot)$ and $\widehat{G}_{\Xi|X,\widehat{V}}(\cdot)$ are the estimates of conditional survival functions of the variables τ and Ξ, respectively. Consequently, an estimator of the associated conditional density function can be constructed as

$$\widehat{o}_{\tau|X}(t,x) = \int \widehat{f}_{\tau|X,\widehat{V}}(t|x,\widehat{v})(d\widehat{v}) \int \widehat{G}_{\Xi|X,\widehat{V}}(\xi|x,\widehat{v})(d\widehat{v}), \tag{4.24}$$

where $\widehat{f}_{\tau|X,\widehat{V}}(t|x,\widehat{v})$ is the estimate of the density function of τ. Finally, an estimator of the HSF is given by

$$\widehat{\lambda}_\tau(t,x) = \frac{\widehat{o}_{\tau|X}(t,x)}{\widehat{J}_{\tau|X}(t,x)}. \tag{4.25}$$

4.5 Application

The duration of an interbirth interval can be affected by a variety of parity-specific factors, other than contraception efficiency. These factors include, among others, breastfeeding duration, temporary postpartum infecundity and the characteristics and survival of the preceding child. For this reason, we choose to assess the net effect

of contraception fertility outcomes via its effect on the length of the duration to first pregnancy. The choice of waiting times to first conception is also justified by the fact that the age at which childbearing begins is not only an important determinant of the overall level of fertility, but is also a key factor in the realized level of a woman's human capital investment (*i.e.* education and work experience).

4.5.1 Data

In what follows, we estimate our model using information on mothers at first conception from the DRC's 2007 Demographic and Health Survey (DHS). We define the time to first pregnancy to be the difference between the age of the woman at first conception and the age at which she had her first intercourse. In the analysis, we drop virgin women from the sample, and consider those who are sexually active, but still childless, to be right-censored observations.

According to our theoretical model, contraception efficiency is the main covariate that influences the latent reproductive health process. In the data set, contraception methods are classified as *traditional*, *folklore* and *modern*. While the survey distinctly differentiates between traditional and modern methods, the definition of what will constitute a folkloric contraception method is not clear. The traditional contraception methods generally include less efficient natural contraception techniques, such as period abstinence (also known as rhythm), withdrawal and abstinence.

Modern methods refer to relatively efficient contraception methods, which involve the use of objects external to the woman's body that can help with birth control. These include pills, intrauterine devices (IUD), injections, diaphragms, condoms, sterilization, implants, foams/jellies. But a natural process, such as lactational amenorrhea is also classified as one of the modern methods. Any method not specifically

mentioned above is considered to be a folkloric method, as long as it is believed to be less efficient than any of the traditional methods. In recording the information, if a woman reports having used more than one method, the most efficient takes priority, and is recorded against her name to the exclusion of the less efficient technique. In the same vein, a woman is recorded to have used a traditional method if she has used both a traditional method and folkloric method.

The other covariate of interest for our analysis is the initial value of the stock of reproductive health capital. Although it is unobserved, we assume that this initial value is a function of socio-economic factors, such as the woman's labour market outcomes, her family structure, the level of poverty in her immediate childhood environment and the availability of child related transfers. Since the DRC lacks a formal social welfare system, we let the number of the woman's older siblings represent the value of the child-related support she can expect to access at childbirth.

Summary statistics in Table 4.1 suggest that more than 40% of women in the sample are classified as poor, live in small towns, and never used modern contraception methods in their lives. They also indicate that the average woman has at least 2 older siblings, first got married at the age of 18, and have a duration of 2 years and 9 months from the time of first intercourse to first childbirth.

4.5.2 Estimation Results

The estimated empirical results – see Table 4.2 – are consistent with our theoretical model, and suggest that demand for contraception services is negatively correlated with the rate at which the childbirth risk process approaches the barrier at zero. In other words, higher contraceptive expenditure leads to a longer waiting period from the first intercourse to the birth of the first child. We also note that earnings and

Table 4.1: Summary Statistics

Nominal Variables				
	Levels	%	∑%	*Obs.*
Childhood Residence	Capital	15.33	15.33	998
	City	18.94	34.27	1,233
	Town	65.73	100.00	4,280
Wealth Status	Poorest	21.12	21.12	1,375
	Poorer	19.52	40.64	1,271
	Middle	19.46	60.10	1,267
	Richer	19.80	79.90	1,289
	Richest	20.10	100.00	1,309
Contraception Use	Never	46.05	46.05	2,998
	Folkloric Only	01.00	47.04	65
	Traditional Only	30.95	77.99	2,015
	Modern Only	22.01	100.00	1,433
Continuous Variables				
	Min.	Median	Mean	Max.
Survival Time (Years)	0.58	2.75	4.08	35.83
Unmet Need (%)	0.00	16.13	17.40	48.39
Age at first Marriage (Years)	10.00	18.00	18.29	45.00
Number of Older Siblings	0.00	2.00	2.77	15.00

Sample size, $N =$ 6,511.

Source: 2007 DHS DR Congo.

Table 4.2: Threshold Regression Cure Rate Model Estimates

AIC	Model 1		Model 2	
	27,738		27,619.74	
	Estimate	Std. Dev	Estimate	Std. Dev
$\ln w_0$				
Intercept	.03773	.03726	.09798	.03763
Age at First Marriage	.03379	.00168	.03126	.00179
Childhood Residence	.03144	.01052	.03752	.01611
Contraception Choice	.00946	.00608	.01506	.00605
μ				
Intercept	1.26853	.05809	1.19412	.27438
Wealth	.01097	.00468	.01078	.00509
Number of older Siblings	.00498	.00251	.00588	.00276
Log Contraception Demand	-.19381	.01608	-.16735	.06230
logit c				
Intercept			7.81133	.53863
Age at First Marriage			-.19923	.02040
Contraception Choice			.630267	.19064

the number of older siblings are positively related to the drift, which suggests, as expected, that the availability of resources accelerate the rate at which the childbirth risk process approaches the threshold. On the other hand, as expressed by the positive sign of the estimates linked to age at first marriage, childhood residence, and choice of contraception method, women who use more efficient contraception methods start the spell with a lower risk of giving birth to the first child. The same also applies to those women, who spent their childhood years in a small town or those who first got married at a relatively advanced age.

4.5.2.1 Estimated Hazards and Probabilities of First Childbirth

As expected from our theoretical model, the shapes of the estimated hazards and cumulative hazards suggest non-proportionality. To show this, we group the women into three different risk groups: *High*, *Moderate* and *Low*. Using $\hat{w}_0$, the predicted value of the initial childbirth risk status, we define a high-risk group as the one including women that are closer to the point of absorption than the moderate or the low-risk group. Specifically, we assume that a woman belongs to a *high* risk group if her predicted value of the initial value of the stochastic process is less than the *lower* quartile of $\hat{w}_0$, *moderate* risk if it is between the lower and the upper quartiles, and *low* risk if it is above the upper quartile.

The graph in on the left panel of Figure 4.3 suggests that women in the low-risk group start their reproductive history with a *wait and see* attitude and have higher expectations of the contraception related benefits than the other groups. Using the median by risk group of the individual estimated values of the parameters w_0 and μ, we are able to show that the average low-risk woman is characterized by a delay in hazard, before catching up with the hazards of any average woman in other groups with higher risk. This confirms a stylized fact of a delay in hazard for low-risk groups that has been reported by other scholars (see Aalen et al., 2008, p. 414). As a consequence, the probability of the onset of motherhood by any given time is clearly lower for those women who start their active sexual life with higher expectations of contraception related benefits, than for those women who start with lower levels of the initial value of the expected future benefits linked to contraception.

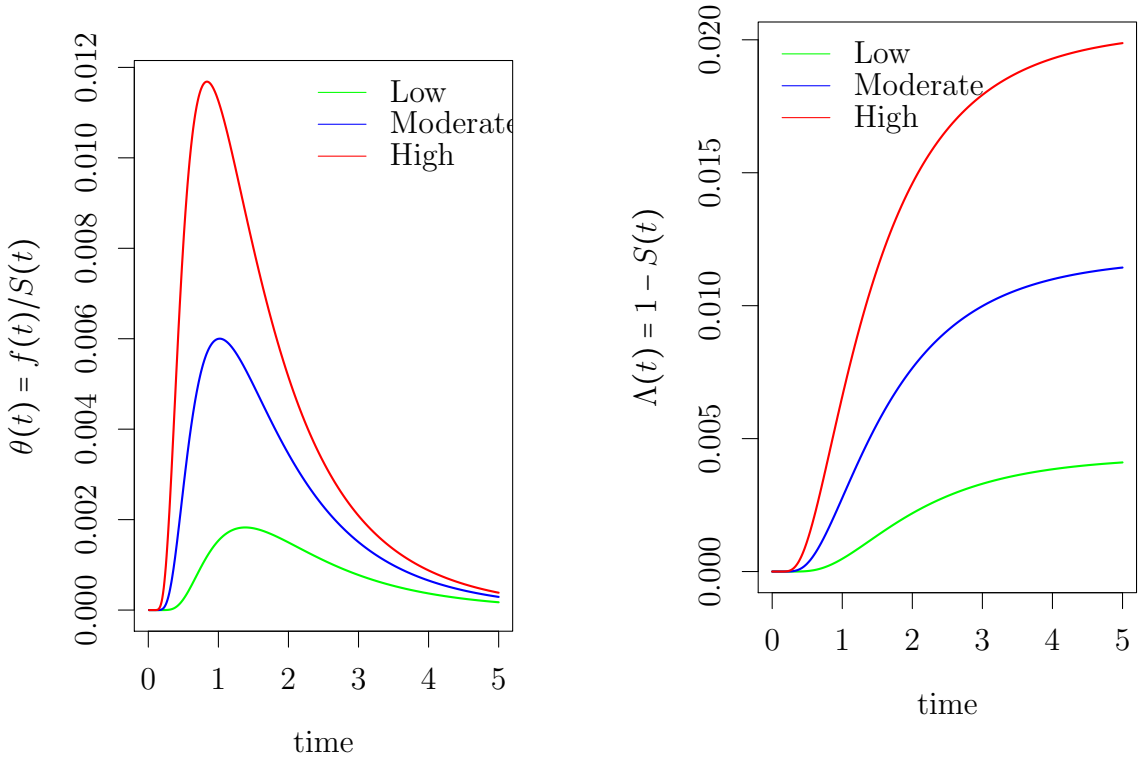

Figure 4.3: *Hazards* (left panel) and the corresponding *Probability of Giving Birth by Time t* (right panel) by *Risk Group* according to the estimated *Model 2*.

4.6 Conclusion

The aim of this chapter was to present evidence on the negative relationship between contraception efficiency and the timing of childbirths and evaluate the return to contraception. The relationship is of importance, because of the presumption that optimal birth timing, and ultimately optimal family size, is achieved through the practice of birth control. This finding can be used as a building building block to a possible explanation of the puzzling negative relationship between income and family size. We presented a model for birth timing and focused our empirical analysis on

the duration to first childbirth using data from a high fertility country. Our empirical results suggest a negative association between contraception efficiency and the length of the duration to first childbirth. Clearly, the use of higher efficiency modern contraceptive methods result in the postponement of the onset of motherhood.

Chapter 5

Conclusion

As noted throughout this thesis, the interaction between fertility and measures of economic development is closely related to the literature on the demographic transition. There are suggestions from the demographic literature that fertility has been reduced by the adoption of family planning services during demographic transitions (Coale, 1984). According to Coale (1984), three prerequisites should come to prevail for there to be a sustained reduction in fertility caused by the use of contraception: (i) fertility must be within the calculus of conscious choice leading to an optimal completed family size; (ii) effective techniques of fertility reduction must be accessible; and (iii) reduced fertility must be viewed as advantageous. However, based on the central assumption that the fundamental force behind fertility transition is the reduced parental demand for children brought about mainly by the changing opportunity costs of childbearing, current demand theories of the fertility transition only cover the first and third factors. This implies little to no attention being paid to the impact of contraception knowledge and availability.

Our approach unites all three of Coale's prerequisites for fertility decline by recasting the fertility-income debate into the income-contraception-fertility space. We

argue that the puzzling negative effect of income on fertility is due to the lack of an explicit model of the demand for contraceptive efficiency within the current economic theories of fertility. Although a woman's probability of conception depends on external biological factors, actual fertility outcomes are stochastically explained by family planning choices and sexual behaviour. The biggest intellectual challenge then becomes the analysis of the determinants of the demand for family planning services and the subsequent impact of the latter on fertility.

The hypothesis is set forth that the interaction between a woman's fertility outcomes and her living standard is mediated by her contraceptive behaviour. If one considers contraceptive use to be the result of a conscious decision-making process, and a deliberate purposeful action on the part of a woman/couple, it implies that demand for family planning would provide a channel of choice, if not the only one, through which economic factors such as income would be expected to explain observed fertility outcomes. In this setting, a rather interesting research theme revolves around the characterization of the way in which a woman/couple's contraception strategy evolves over its life cycle as a function of economic variables, such as the time paths of family resources, the parent's opportunity cost of time, the cost of contraception (see Heckman and Willis, 1976) and how this contraceptive behaviour, in turn, affects fertility outcomes.

We develop a fertility model that combines the literature on the human capital theory of the demand for health (see Grossman, 1972, 2000; Becker, 2007), with fertility transition theories (see Coale, 1984; Cleland and Wilson, 1987). The principal contribution of this study is to the consideration of family planning as an investment in reproductive health capital and its relation to women's labour market outcomes. In this framework, it is implied that contraception has an impact on both the standard

of living and fertility outcomes. More specifically, there is a fundamental difference between health capital and other forms of human capital. While a person's years and quality of schooling and training may affect her productivity, good health can be viewed as a durable capital stock that determines the amount of time the person spends being productive (Schultz, 1961; Grossman, 1972; Strauss and Thomas, 1998). Given the fact that in the large majority of communities the burden of childbearing rests disproportionately on women, investment in family planning can also be considered to be an investment in a female-specific form of human capital. Conceptually, we assume that women with positive reproductive health outcomes are more productive, for longer, in the labour market.

So, in general, one should expect access to reproductive health services to be related to participation or performance in the labour market. Reproductive health services encompass many areas, such as maternal health services and services related to sexually transmitted infections, but the analysis herein focuses on access to family planning services. In particular, delaying childbearing through birth control allows a woman to achieve higher levels of education and improves her time of successful activity in the labour market. Consequently, through contraceptive practice, a woman's fertility outcome is at least partially a result of conscious decision-making and a deliberate purposeful action, which provides researchers which a channel of choice to analyze the impact of economic variables on family-making decisions.

In what has transpired, we contend that for a woman participating in the labour force, the main objective of the investment in reproductive health is to avert any adverse effect on the accumulation of human capital that may arise due to mistimed, risky or unwanted childbearing. A pure reproductive health investment model would then suggest that a woman's optimal investment in family planning is driven by her

desire to maximize her lifetime net money income production function, subject to the dynamics in the her own stock of reproductive health capital (see Ben-Porath, 1967). In this context, higher wages would induce investment in more efficient family planning services.

In this triangular framework, the role of family planning as an intermediate variable in the causal path between income and fertility is investigated using causal mediation analysis (see Baron and Kenny, 1986; Pearl, 2001, 2012*b*; Imai, Keele and Yamamoto, 2010). Since competing scientific theories often imply that different causal paths underlie the same cause-effect relationship, the causal mediation analysis has the advantage of going beyond simple causal effects to provide the driving causal mechanisms (Imai, Keele and Yamamoto, 2010). We illustrate the estimation of certain aspects of our models using data from South Africa and the Democratic Republic of Congo.

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