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Responsiveness of Child Health to Maternal Fertility in Central African Republic

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Abstract

This paper investigates the effect of fertility on child health status, using data from the sixth edition of the Multiple Indicator Cluster Survey (MICS6) of the Central African Republic (CAR) collected in 2018 by the government's statistics office. Specifically, the paper seeks: to assess the direct and indirect effects of maternal fertility on child health. To achieve these objectives, we use a framework based on the quantity-quality theory of the new household economy and the control function modeling strategy—which simultaneously corrects for endogeneity, sample selectivity and unobserved heterogeneity biases. Results show that, having an additional child significantly increases the risk of sickness in children under 5 years in CAR. Results also show a positive and highly significant indirect effect of fertility on sickness as depicted by the interaction of fertility and its residual—a clear indication of undesirable complementarities between fertility and unobserved correlates of fertility in widening sickness probability among children. These findings suggest that public intervention that improves spending on social services, such as education, preventive child health and family planning programmes, would reduce fertility, and initiate the process of better child health and productivity in adulthood.

Keywords

Maternal Fertility, Child Health, Control Function Modeling, MICS, CAR

1. Introduction

Microeconomic analysis of household fertility behaviour highlights the idea that households with a higher number of children devote fewer resources to each child, precisely the more children a household has, the fewer resources the household can allocate to each child and thus the quality of each child will be lower (Maitra

& Pal, 2008; Baye & Sitan, 2016). Conceptually, Becker & Lewis (1973) formulated the problem as a trade-off between the number of children and investments to improve their quality. In this context, children with many siblings have lower human capital indicators than children from smaller families. Thus, children in large families are disadvantaged by having to share material resources, as well as the time and attention of their caregivers, with other siblings (Glick et al., 2007). Children with many siblings generally present worse indicators of long-term nutritional status, such as height-for-age z-scores (HAZ), which reflect stunting (Wolfe & Behrman, 1982; Lalou & Mbacke 1992; Desai et al., 1993), lower school performance (Blake, 1981; Lloyd, 1994) and lower enrolment rates (Rosenzweig & Schultz, 1987; Alderman et al., 2001).

In this context, several studies carried out in developing countries show a negative association between nutritional indicators and family size or fertility (Heller & Drake, 1979; Wolfe & Behrman, 1982; Horton, 1986; Alderman & Mundial, 1990; Lalou & Mbacke, 1992; Desai et al., 1993). Large family size is also often associated with lower schooling or educational attainment of children (Birdsall, 1980; Blake, 1981; Jamison & Lockheed, 1987; Basu, 1994; Lloyd, 1994). The standard interpretation of this relationship is that an additional child increases the demand for resources in households that face fixed constraints on financial resources and parental time (Tray, 1973; Horton, 1986). Other studies tend to assume that the composition of the child population classified by health status is not related to past fertility decisions (Makepeace & Pal, 2008). Moreover, poor rural households with access to physical assets may not care about current income and thus choose to have more children as insurance against anticipated future income declines.

Since there appears to be lack of consensus on the causal relationship between high fertility and child health in the existing literature, it is interesting to empirically verify this association in the context of CAR. Thus, a key research question arises: What are the direct and indirect effects of fertility on the child health status of under 5 years in the Central African Republic? The rest of the paper is organised as follows: Section 2 presents the key indicators of maternal and child health in the Central African Republic (CAR). Section 3 reviews the relevant literature; Section 4 presents the methodology and the data. Section 5 presents the results and discussions and Section 6 concludes the study and addresses the policy implications.

2. Maternal and Child Health Indicators in the Central African Republic

The Central African Republic, like other developing countries, has adopted the Sustainable Development Goals for health in the period 2015-2030 to improve the health of Central Africans by reducing maternal mortality, under-five mortality with a focus on reducing neonatal mortality, controlling HIV and other pandemics in combination with the other goals. Since the 2000s, a number of reforms have been introduced to improve health service delivery and facilitate

the development of the private health sector.

In 2016 the authorities adopted a comprehensive and ambitious health strategy. The main objectives, as defined in the National Health Development Plan (PNDS), are to contribute to the improvement of the health of the population by ensuring quality primary health care for the entire population, particularly in a context of poverty and armed conflict. The main strategic orientations are six in number, including: 1) rehabilitating and developing health infrastructures, 2) increasing the availability and quality of essential care, 3) reducing infant and maternal mortality, and 4) halting the spread of endemic diseases, particularly HIV/AIDS, 5) strengthening the health system, 6) promoting an environment conducive to health, including aspects of health, sustainable development, and emergency preparation and response.

Despite these efforts, analysis of the available indicators suggests that CAR will struggle to meet the MDG targets by 2030. The infant mortality rate is 76.47 deaths per 1000 live births, a considerable gap compared to the African average of 50.3 deaths per 1000 live births, whereas the MDGs aim to reduce this to less than 25 deaths per 1000 births. The maternal mortality ratio is 820 deaths per 100,000 live births compared to the African average of 243 deaths per 100,000 births and the MDG target of 70 deaths by 2030. Similarly, only 22% of the population had access to quality health care compared to the African average of 38%. The HIV prevalence rate was 6.8% among pregnant women, while the MDGs aimed to halt its spread.

Furthermore, the descriptive statistics of the MICS surveys indicate that between 2006 and 2018, the prevalence of stunting among children under five increased from 18.7% to 39.8%, acute malnutrition or wasting increased from 2.3% to 9.4%, underweight children from 8.1% to 23.5%, diarrhea from 19% to 38%, pneumonia from 7% to 8.2% and malaria from 20% to 40%, while the MDGs to which CAR has subscribed aim to end all forms of malnutrition by 2030.In addition, fertility remains relatively high in CAR, with the average number of children a woman in her late fertile years can have being 6.4% children. This context makes CAR and interesting case study to investigate the nexus between fertility and under 5 health status.

3. Literature Review

The theory of household production developed by Becker (1965) allows for the analysis of the rational behaviour of households, which is a behaviour based on the maximisation of their utility. This rational behaviour is constructed using the new household economic framework, which is a model combining education, marriage/birth choices and labour supply (Cigno, 1991). Becker tries to explain this rational behaviour in terms of the satisfaction that each choice (having a child) brings to the household. As such, the choice of fertility over a lifetime emphasises different facets of this decision process, but the trade-off between the quantity of children and their quality (education and health) has become a cen-

tral element (Becker & Lewis, 1973; Mincer, 1963; Schultz, 1986).

Blake's (1981) contribution of the dilution theory to the quantity-quality theory makes it possible to clearly define the link between sibling size and the human capital outcomes of children. According to this theory, lower fertility leads to the distribution of family resources among fewer children, which increases the resources available for each child and improves the outcomes of each child.

But this notion of child quality has been controversial, both in terms of the concept itself and in terms of the assumption that all children in the same family have the same quality. Moreover, the assumption of the same quality for children in the same family has been refuted by empirical observation. It was recognised that parents are not free to choose any level of quality. They are constrained by their own education and standard of living or quality (Macunovich, 2003).

The main limitation is that the dilution theory does not take into account the distribution of resources within the household. Even when families have fewer children, the potential for intra-household discrimination means that not all children necessarily benefit equally (Alderman et al., 2001). Moreover, even if the total fertility rate is falling, the number of children in households may remain stable or even increase. This can happen when fertility decline is concentrated in certain groups (Giroux, 2008) or when the prevalence of foster care increases and additional foster children compete with existing children for limited resources, especially in sub-Saharan Africa.

Lloyd (1994) and Desai et al. (1993), demonstrate that the relationship between family size and child outcomes, is likely to depend on the context of family culture, the subsidy of child-rearing costs and the stage of demographic transition. Shapiro & Tambashe (2001) show that fertility decline in rural areas lags far behind that in urban centres, while Kirk & Pillet (1998) found substantial variations in fertility decline by family socio-economic status.

Desai et al. (1993) also found that the relationship between sibling size and height for age depends strongly on the extent to which parents bear the cost of child welfare. Thus, even if similar declines in fertility occur in all regions, this does not necessarily translate into a reduction in stunting in all regions. On the contrary, the influence of these factors may increase or decrease the dividend as proposed by the dilution theory.

According to Thirlwall & Keynes (1999), there are a number of reasons why maternal education should reduce fertility: 1) education improves work opportunities for mothers, which makes it more time-consuming to have children; 2) educated mothers want their own children to be educated, which increases the cost of having children; 3) education and literacy make women more receptive to information about contraception; 4) education and employment delay marriages and the time available to raise children; and 5) education improves women's status, bargaining power and independence, encouraging and enabling them

to make more rational choices[1].

Most evidence from developing countries suggests that having more siblings is disadvantageous for a child's well-being in terms of education and health. Lalou and Mbacke (1992) found that having more siblings increased the likelihood of children suffering from malnutrition in Mali.

Empirical studies on the effects of fertility on child health are few in developing countries and particularly in Sub-Saharan Africa. These few studies sought to analyse the influence of fertility on child health status. For example,

For example, Baye and Sitan (2016) used the 2004 Cameroon Demographic and Health Survey to highlight the effects of maternal fertility choices on child health status captured by the short-term anthropometric indicator, weight-for-height z-scores. They used the control function approach to address the endogeneity problem, sample selection bias and unobserved heterogeneity bias of fertility choices. The results showed that women with twins have higher fertility and that maternal fertility has a negative and significant effect on child health.

In the same spirit, Kabubo-Mariara, Mwabu and Ndeng'e (2009) using data from the 2003 Kenya Demographic and Health Survey, investigate the impact of fertility on child health as captured by the infant mortality indicator. They used the instrumental variables method and the control function approach to take into account the problem of endogeneity of fertility and complementarity arising from unobservable determinants of child health, which may be correlated with fertility. The results suggest that an increase in fertility by one additional child increases the probability of mortality by about 1%. With instrumentation, an increase in fertility by one child increases the probability of mortality by 6% in rural areas, but surprisingly reduces it by 4% in urban areas. The coefficient in urban areas, however, is insignificant.

To the best of in our knowledge, no study has examined the nature of the link between fertility and child health in CAR. In order to contribute to this growing literature on child health status, this study builds on existing work and contributes to the literature on child health and women's reproductive health in CAR.

4. Theorical Framework, Methodology and Data

4.1. Theorical Framework

We consider child health/nutrition through the behaviour of the mother's demand for reproductive health services by considering a framework in which the mother's utility function includes child health. The underlying theory guiding this framework is the New Household Economy (NHE) model of the family, which recognises that households also derive utility from goods and services produced at home or for which there is no market.

The application of this theoretical framework to child health is well known, and is discussed in detail by Behrman and Deolalikar (1988). In a simple version

[1]Thus, from a vicious circle of no education, high fertility, poor child health and low productivity. Women's education also leads to a virtuous circle of lower fertility, better childcare, more educational opportunities and higher productivity.

of the theoretical framework, the mother is generally seen as maximising a utility function defined over leisure, market and home-produced goods such as child health and child health-related goods such as family planning services, and is faced with three main constraints: a budget constraint, a time constraint and a health production function. The health production function will depend on market inputs such as food (or nutriments) and health services, the mother's time and characteristics, environmental and community characteristics such as access and proximity to public goods, and the child's and father's endowments. The empirical link between fertility choices and children's health and the factors that condition this link can be used to derive public policies that would encourage optimal sibling size and improvements in the human capital (health and education) of children.

4.2. Methodology

Empirical model

Fertility choices are assumed to be an important input into the production of child health. Since child health (CH_i) and fertility (Fe) are jointly and simultaneously determined and each has a behavioural interpretation ceteris paribus, their underlying links can be described by the following structural equations (Baye, 2010; Baye & Sitan, 2016):

$$\mathrm{CH}_i^* = \varphi_0 + \varphi_1 \mathrm{Fe}_i + \sum_{k=2}^{n} \varphi_k X_{ki} + \varepsilon_1 \tag{1}$$

$$\mathrm{CH}_i = \begin{cases} 1 \text{ if } \mathrm{CH}_i^* > 0 \\ 0 \text{ if } \mathrm{CH}_i^* \le 0 \end{cases}, \text{ when HAZ} < -2, \mathrm{CH}^* > 0; \text{ when HAZ} \ge -2, \mathrm{CH}^* \le 0$$

$$\mathrm{Fe}_i = n_0 + n_1 h_i + \sum_{k=2}^{n} \eta_k X_{ki} + \varepsilon_2 \tag{2}$$

where CH_i^* is a latent variable that denotes the probability that a child i is sick—that is, when HAZ < −2 and Fe_i denotes fertility. X_i is a set of exogenous variables affecting child health such as household, parental, child and environmental characteristics, φ_i are structural parameters of the child health production function to be estimated, ε_1 the error term. That is independent and identically distributed.

Equation (2) is the linear projection of the endogenous explanatory variable, Fe expressed as a function of all the exogenous variables including, h_i, is the vector of variables excluded in the structural equation, the instruments plus the exogenous variables in the structural equation of sickness (Equation (1)) and η_i is the coefficient of associated variables. These instrumental variables have direct effects on fertility, contraceptive use and the interaction between contraceptive use and fertility but do not have direct influences on the child health production function, except through maternal fertility. X_i is the vector of variables included in the structural equation. These are variables that directly explain the child health production function and ε_2 is the error term.

To correctly estimate the parameters of our model, it is important that the effects of endogenous fertility and the sample selection indicator on child health are identified. In our case, endogeneity is suspected as soon as fertility is subject to household preferences/choices. That is, as soon as the health of the children and the level of fertility are jointly determined, then there is a possibility of causality. Moreover, many studies have shown that maternal fertility is a key determinant of child health outcomes (Preston, 1975; Case & Paxson, 2001). To address the endogeneity problem, the strategy is to use the instrumental variables (IV) method[2]. In this case, potential instruments for fertility are needed to estimate the effects of fertility on child health in a consistent way. Fertility instruments are those factors that affect fertility choices without directly influencing child health.

Furthermore, the fact that health inputs are choice variables introduces unobserved heterogeneity in the child health production function. Failure to account for this type of heterogeneity will lead to unreliable estimates and may mask a significant impact on child health of early prenatal medical care (Rosenzweig & Schultz, 1987; Mwabu, 2009). In addition, the estimates may not be applicable to all children aged 0 - 5 years because of the potential selection of the child health sample, which only includes children whose heights or weights were not collected. Therefore, excluding these children from Equation (1) makes our estimation sample non-random.

To simultaneously account for potential endogeneity, non-linear interactions between unobservable variables and explanatory variables (repressors) of children's health and sample selection bias, Equation (1) is augmented to Equation (3) and can be rewritten as: a control function as follows (Garen, 1984; Mwabu, 2009; Baye & Fambon, 2010; Wooldridge, 2015):

$$\mathrm{CH}_i^* = a_0 + a_1\mathrm{Fe}_i + \sum_{k=2}^{n} a_2 X_i + \beta_1\widehat{\varepsilon_{fe}} + \beta_2\left(\mathrm{Fe}_i * \widehat{\varepsilon_{fe}}\right) + \lambda\mathrm{IMR} + v \qquad (3)$$

where $\widehat{\varepsilon_{fe}}$ is the fertility residual derived after estimation of the reduced form fertility model (Equation (2)) and serves as a control for unobservable variables that are correlated with (Fe_i), thus allowing the endogenous variable to be treated as if it were an exogenous covariate during estimation, $\left(\mathrm{Fe}_i * \widehat{\varepsilon_{fe}}\right)$ is the interaction term of fertility with its residual, which controls for the indirect effect of fertility on child health status attributable to the non-linear unobserved heterogeneity associated with fertility; IMR is the inverse of the Mills ratio (the pseudo-error term) derived after estimation of the probit for sample selection, holds constant the effects of sample non-randomness on the structural parameters in the usual framework and v is the error term of the estimating equation; a, β and λ are vector of parameters to be estimated. $\widehat{\varepsilon_{fe}}$, $\left(\mathrm{Fe}_i * \widehat{\varepsilon_{fe}}\right)$, and IMR are the control function variables.

Since the nature of the dependent variable is qualitative and binary, the struc-

[2]In general, acceptable instruments should be relevant: if their effect on the potentially endogenous explanatory variable is statistically significant; strong: if the effect size is sufficiently large; and valid: if they are uncorrelated with the structural error term and exclusion restrictions are properly excluded from the estimating equation. Valid instruments are generally difficult to find.

tural equation is estimated as a Probit model controlling for endogeneity, sample selection and unobserved heterogeneity biases.

In essence, Equation (3) is subject to a number of important conditions regarding specification, estimation and model testing: 1) the standard t-statistics and F-statistics are applicable to Equation (3); 2) if β_1, β_2 and λ are statistically equal to zero, the structural parameters of the child health equation can be estimated consistently by the simple Probit using the selected sample; 3) if β_2 and λ are statistically insignificant, the control function variables in the equation are only the fertility residuals predicted from the reduced form equation. In this case, the structural parameters can be consistently estimated by the IVProbit on the selected sample; 4) if only λ is statistically equal to zero, equation 3 can be estimated by omitting the IMR, 5) if λ is statistically different from zero, the estimation of the resultant equation will be done through the control function approach to take into account the sample selection bias; and (6) if β_1, β_2 and λ are all statistically significant, the control function approach is preferred.

4.3. Presentation of Data and Model Identification

Presentation of data

The data used in this study come from the sixth edition of the Multiple Indicator Cluster Survey (MICS6) conducted in 2018 by the Central African Institute of Statistics and Economic and Social Studies (ICASEES), as part of the global MICS survey programme. During the survey, 11,000 households were registered and 9778 eligible women aged 15 - 49 were identified. Of the eligible women, 9202 were successfully interviewed. Our unit of analysis is the child, and a total of 8921 children aged 0 - 59 months were recorded in the survey.

Model identification

The correlation between fertility and child health does not necessarily indicate causality unless a convincing cross-effect identification strategy can be developed (Schultz et al., 2006). Since fertility and child health are jointly determined, exogenous variations in fertility are required. To correctly estimate the parameters of our model, it is important that the effects on child health of endogenous fertility and the sample selection indicator are identified. In this case, the identification requires at least two exclusion restrictions because there are two equations that need to be solved simultaneously. That is, we need at least one exogenous instrument for endogenous fertility and another exogenous variable that determines the selection of children in the estimation sample.

In this regard, we use two instruments[3], captured at the community level (primary sampling unit, PSU), where the woman and child live in order to avoid weak instruments, in the same way as Delprato et al. (2015). The instrument is the non-self-cluster-mean[4] or the neighbourhood average of fertility and the sample selection indicator, they measure the average value of fertility share of

[3]Following the advice of Baye and Sitan (2016), our instruments are captured at the neighbourhood level where the woman and child live.

[4]"the non-self-cluster-mean" a community variable calculated excluding the woman to whom it is attributed.

women living around a woman disregarding of her own fertility and the proportion children living around a child disregarding the child under consideration.

In this context, the decisions of other groups of individuals in the neighbourhood or in society can affect an individual's decision (De Grange et al., 2015). Specifically, fertility and selection into the sample can be influenced by the average fertility pattern of other women at the community level. For example, when a large number of women in a community do not control their fertility, this is likely to increase a woman's fertility and when a large number of children in the community are selected, this is likely to increase the likelihood of a child being selected. Meanwhile, the neighbourhood average (fertility) and proportions (selection) are not expected to correlate with a given child's health status fertility and selection.

5. Empirical Results and Discussion

5.1. Analysis of Descriptive Statistics

Table 1 summarises the statistics describing the variables of interest. 38.3% of the children aged 0 - 5 years suffer from stunting (acute malnutrition). The average mother is 28 years old and has an average of 5.36 children. The average age at first marriage is 16.71 years. 38.7% of the women have no education, 55.4% have primary education and 5.9% have secondary education and above. About 93.1% of mothers are underweight and 5.1% are of normal weight, which may justify the high rate of malnutrition among children. As for the wealth of the households in our sample, 20.2% are poor and 21% are rich. 64.1% are from rural areas and 35.9% from urban areas. This may explain the relatively high maternal fertility rate per mother.

5.2. Estimation of Sample Selection and Reduced Form

Table 2 (Column 2) presents the parameter estimates of the probit model, of the probability of including a child in the estimation sample, the result show that the average selection of other children at the neighbourhood level is significant at the 1% level, which means that our instrument is relevant. Wald tests performed on our instrument yield significant results. This result indicates that our instrument is statistically relevant and we can conclude that the selection of children entered at the neighbourhood level is valid. Therefore, the selection of the remaining children captured at the neighbourhood level increases the probability of selection into the sample. Thus, this average for the variables included in the other children selection equation increases the probability that a child is selected into the sample by 32.5%. For the variables included in the outcome equation, being a girl and having secondary education and for the mother tend to increase the probability that a child will be selected in the sample.

5.3. Reduced form Equation and Sample Selection Estimates

Table 2 provides in the column 3 the results of the OLS estimation of the reduced

Table 1. Descriptive statistics.

Variables	Mean	Std. Dev.	min	max
Dependent variable				
Children with long-term malnutrition	0.209	0.407	0	1
Children without malnutrition	0.791	0.407	0	1
Endogenous explanatory variables				
Maternal fertility = number of children born to a woman	5.357	2.133	2	14
Exogenous explanatory variables				
Age of the child	29.32	17.324	0	59
Age of child squared	11.598	10.434	0	34.81
Male	0.498	0.5	0	1
Girl	0.502	0.5	0	1
Child with diarrhea	1.795	0.169	1.167	2.944
Age of mother	28.072	9.163	15	49
Age of mother squared	8.72	5.605	2.25	24.01
Mother without education	0.387	0.487	0	1
Mother with primary education	0.554	0.497	0	1
Mother with secondary education and more	0.059	0.235	0	1
Mother with low weight	0.931	0.254	0	1
Mother with normal weight	0.051	0.221	0	1
Poor household	0.202	0.401	0	1
Rich household	0.21	0.407	0	1
Urban area	0.359	0.48	0	1
Rural area	0.641	0.48	0	1
Instrumental variables				
Non-self-cluster mean of fertility	5.354	0.525	3.429	7
Non-self-cluster mean of selection	0.965	0.061	0	1
Simple selection variable	0.965	0.183	0	1
Control function variables				
Residual of fertility	5.363	0.846	2.972	8.658
Fertility residual * fertility	29.474	14.128	5.944	103.006
Inverse Mills ratio	0.38	0.2510202	0.0015	1.282

Note: Source: Computed by a Authors from MICS-6_RCA.

form fertility equation. The estimation of the reduce form show that the average fertility of other women entered at the neighbourhood level is significant and positively correlated with fertility at the 1% level. In addition, and the significance of the Fisher statistic reveals that the selected instrument is valid and proof

Table 2. Probit estimation of sample selection and the reduced form estimates.

Variables	Child age reported age = 1 and 0 otherwise		Maternal fertility
	Coefficients (1)	Marginal effects (2)	Coefficients (3)
Age of the child	0.009	0.0004	0.014**
	(0.008)	(0.0004)	(0.006)
Age of the square child	−0.015	−0.001	−0.019**
	(0.013)	(0.001)	(0.009)
Girl	0.135**	0.007**	−0.042
	(0.068)	(0.003)	(0.049)
Child with diarrhoea	−0.117	−0.006	0.081
	(0.199)	(0.010)	(0.144)
Age of the mother	−0.013	−0.001	0.499***
	(0.031)	(0.002)	(0.023)
Mother's age squared	0.024	0.001	−0.773***
	(0.046)	(0.002)	(0.034)
Mother with primary education	−0.025	−0.001	−0.083
	(0.073)	(0.004)	(0.052)
Mother with secondary education and above	0.393*	0.014**	0.146
	(0.226)	(0.005)	(0.136)
Normal mother's weight	−0.169	−0.010	−0.029
	(0.134)	(0.009)	(0.111)
	(0.0771)	(0.004)	(0.057)
Average household	−0.082	−0.004	0.0732
	(0.087)	(0.005)	(0.064)
Rich household	0.091	0.004	-0.0186
	(0.091)	(0.004)	(0.063)
Rural areas	0.003	0.0001	0.076
	(0.084)	(0.004)	(0.060)
Non-Self-Cluster mean selection	6.569***	0.325***	
	(0.373)	(0.025)	
Non-Self-Cluster mean fertility			0.810***
			(0.047)
Constant	−3.796***		6.439***
	(0.683)		(0.540)
Number of observation	8921		8140
Log pseudolikelihood	−754.38038		
Predicted probability of the model	0.97		
R^2/Pseudo-R^2	0.1637		0.230
F-stat [df; *p*-value]/wald chi^2 [dl-*p*-value]	333.92 [12; 0.0000]		102.86 [12; 0.0000]

Source: Author's calculation from MICS6-RCA data using Stata 14; Note: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$ Robust standard errors in parentheses.

that our model is well fitted. The variables in the outcome equation such as the age of the child and the mother are positively and significantly correlated with having more children. Fertility increases with the age of the child and starts to decline thereafter.

5.4. Determinants of the Production of Sickness (HAZ < −2) among the under-5 in CAR

Table 3 presents estimates of the child health (sickness) production function under different assumptions. In particular, column 1 is a simple Probit estimate of the structural parameters of child health production, Column 2 is the estimate of the structural model parameters that account for potential sample selection bias. Column 3 is an instrumental variable Probit (IV Probit) estimate of the structural parameters accounting for potential endogeneity, Column 4 hosts control function estimates without the interaction term, and Column 5 presents full control function estimates estimate correcting for endogeneity, sample selection and unobserved heterogeneity biases. Columns 1 to 5 show that fertility increases the probability of sickness among the under-5 by 2.4% to 38.8%. This is indication that the magnitude of the effect of fertility on child health depends on the estimation method. To avoid spurious policy implications, it is important to use an appropriate estimation strategy that internalises potential econometric problems.

Table 3. Child health production function under different assumptions.

Variables	Probit model (1)	Heckman selection bias correction (2)	IV probit model with endogeneity correction (3)	Control function model without residual interaction term (4)	Control function model with residual interaction term (5)
Fertility	0.0235***	0.0546***	0.334***	0.3472***	0.388***
	(0.00039)	(0.0007)	(0.0581)	(0.0560)	(0.0546)
Age of the child	0.0143***	0.0327***	0.0518***	0.0423***	0.0576***
	(0.00357)	(0.00457)	(0.0067)	(0.0069)	(0.0072)
Age of the square child	−0.0002***	−0.0005***	−0.0006***	−0.0005***	−0.0006***
	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Girl	−0.058***	−0.056***	−0.064***	−0.064***	−0.137***
	(0.011)	(0.061)	(0.012)	(0.009)	(0.0347)
Child with diarrhoea	0.169***	0.172***	0.148***	0.148***	0.133**
	(0.0392)	(0.0392)	(0.0390)	(0.0383)	(0.0583)
Age of the mother	0.0100	0.0110	0.233	0.233	0.212
	(0.0117)	(0.0117)	(0.0627)	(0.0619)	(0.136)
Mother's age squared	0.001	0.001	0.0002	0.0002	0.0002
	(0.001)	(0.001)	(0.0002)	(0.0002)	(0.0002)

Continued

Mother with primary education	−0.111***	−0.109***	−0.114***	−0.114***	−0.121***
	(0.0362)	(0.0360)	(0.0342)	(0.0338)	(0.0460)
Mother with secondary education and above	−0.450***	−0.431***	−0.431***	−0.431***	−0.495***
	(0.103)	(0.102)	(0.0982)	(0.099)	(0.117)
Normal mother's weight	−0.335***	−0.335***	−0.771***	−0.771***	−0.864***
	(0.0723)	(0.0723)	(0.121)	(0.127)	(0.118)
Average household	0.00353	−0.0477	−0.0154	−0.0154	−0.00598
	(0.0509)	(0.113)	(0.0487)	(0.0457)	(0.0513)
Rich household	−0.187***	−0.186***	−0.201***	−0.201***	−0.233***
	(0.0555)	(0.0551)	(0.0527)	(0.0562)	(0.0588)
Rural areas	0.00279	0.00217	0.0582	0.0582	0.0611
	(0.0481)	(0.0473)	(0.0470)	(0.0470)	(0.0638)
Control function variables					
Fertility residual				0.218**	0.206**
				(0.155)	(0.137)
Fertility multiplied by its residual					0.1661***
					(0.0162)
Inverse Mills ratio		−0.0222***		−0.00123***	−0.0295***
		(0.0045)		(0.0110)	(0.0224)
Diagnostic statistics					
Number of Observations	8441	8441	8441	8441	8441
Pseudo R^2	0.1236				
Wald chi^2 [dl; *p*-value]	255.43 [12; 0.00]	262.33 [13; 0.00]	418.41 [12; 0.00]	261.82 [14; 0.00]	263.57 [15; 0.00]
Rho((ρ)) [robust Standard Error].		−0.989 [0.032]	0.051 [0.0038]	0.071 [0.0019]	0.081 [0.049]
Wald test of indep rho (ρ) = 0 [dl; *p*-value]		1.56 [1; 0.2121]			
Wald exog.chi^2 test [dl; *p*-value]			2.75 [1; 0.0032]	5.22 [1; 0.0022]	5.44 [1; 0.019]

Source: Computed by authors from MICS6-RCA data using Stata 14; Note: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$ Robust standard errors in parentheses.

In this regard, since Column 5 addresses the potential endogeneity, sample selection and unobserved heterogeneity biases, we consider Column 5 results as the preferred estimates. This is especially so because the three control function variables, notably the residual of fertility, the inverse Mills ratio and the interaction of fertility with its residual—accounting for endogeneity, sample selection and unobserved heterogeneity, respectively, are statistically significant at the 1% level. The significance of the control function variables is an indication that using the control function modelling strategy is imperative in the present case study.

The preferred results (Column 5) show that fertility is positively and significantly correlated with the probability of sickness among the under-5 in CAR. More specifically, an additional child increases the probability of long-term sickness among the under five years old children in CAR in the order of 38.8%. This is the direct effect of fertility on child health. This estimate is interesting because the estimated coefficients of the child health production technology under this specification are an improvement to the simple Probit and IVProbit estimates and the control function without the interaction term.

Since the fertility residual and the interaction term (fertility times its residual) are statistically significant at the 1% level, the indication is that maternal fertility is endogenous in the child health production function, and there is evidence of unobserved heterogeneity of responsiveness of child health to maternal fertility decisions. The indirect effect of fertility on child sickness therefore depends on the estimated coefficient of the interaction of fertility with its residual captured at the mean value of the fertility residual. Column 5 shows the indirect effect of fertility on child sickness to be positive and statistically significant at the one present level. This is evidence of undesirable complementarities between fertility an unobserved correlate of fertility in worsening sickness probability among children.

Column 5 also shows the estimated coefficient of the inverse Mills ratio of (−0.030), which is statistically significant at the 1% level. This result supports our choice of the full control function modelling strategy because it can be used to simultaneously purge the structural equation parameters of the three potential econometric problems highlighted earlier. The negative coefficient of the IMR suggests that children selected into the estimation sample are less likely to suffer from sickness than children drawn randomly from the general population. Specifically, since the sample mean of the inverse Mills ratio is 0.38 (**Table 1**), children captured in the estimating sample are 1.14 $(=|-0.030 \times 0.38| \times 100)$ percent less exposed to sickness than their counterparts randomly selected from the general population.

In addition, there is evidence that being a girl reduces the probability of sickness. The pattern of coefficients of child age in all specifications is inverted U-shaped, indicating that younger children are more likely to be sick than their older counterparts. Kabubo-Mariara et al. (2009) for Kenya instead found that younger children are likely to be better nourished than older ones. Mother's weight and education, as well as household wealth unambiguously reduce the probability of sickness among children. As expected, diarrhoea episodes increase the probability of stunting in CAR.

6. Conclusion

This study empirically established a link between maternal fertility and the under-5 child health status using the 2018 CAR MICS-6 data. Specifically, the paper investigated the direct and indirect effects of maternal fertility on child health.

A range of econometric estimation methods was used and the control function approach was found to be the most appropriate strategy, as it simultaneously purges the structural parameters of potential endogeneity, sample selection and unobserved heterogeneity biases. Results showed that controlling for other socioeconomic and demographic factors, fertility significantly increases the risk of sickness among children in CAR. This direct effect was reinforced by the indirect effect as translated by the coefficient of the interaction term (fertility times its residual). This is indication of undesirable complementarities between fertility and unobserved correlate of fertility in worsening the probability of sickness.

These findings suggest that to guide public policy, it is important to expand public spending on social services reaching poor households, such as education for all, preventive child health programs, and family planning—which may accordingly reduce expect fertility and the probability of sickness among children—while initiating the process of improving child health and productivity in adulthood.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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