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Determinants of Cross-Country Child Mortality: A Panel Instrumental Variable Estimation with Endogenous Child Mortality and Fertility

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Abstract: This paper analyses the effects of per capita income, income inequality, female literacy rate, fertility rates and female labour force participation rate on child mortality rate in a panel of 85 countries across the globe for the period 2000-2020. The study uses the panel fixed effects instrumental variable method with the fertility rate as an endogenous variable in the model. The panel IV estimates show a significant negative effect of per capita income and female literacy rate and a strong positive impact of fertility rate on child mortality. But, there is no significant role for income inequality and female labour force participation in controlling child mortality. In countries where there is persistent child mortality, the policies should focus on bringing down the fertility rates through education, especially female literacy and more so on the usage of contraceptive usage. The strong positive relationship between overall economic development and the levels of death rates strengthens the argument that economic development is the best contraceptive. Controlling child mortality rates is an advantage to the population with considerably less pressure on resource use and an even greater advantage to the growth of the economy.

Keywords: Child mortality, fertility, endogeneity, instrumental variable, panel fixed effects

1. Introduction

Child mortality rates such as infant mortality rate and neonatal mortality rate are generally accepted as measures of the health of the population as well as the quality of health care. Though mortality rates refer to the risk of infant deaths, it is widely

used as a crude indicator of the general population's health, the socio-economic status of the community and the level of penetration of quality healthcare services. The child mortality rates are related to other factors that are likely to impact the population's health in general, such as the level of economic development, standards of living, social welfare, morbidity rates and the environment. Child mortality rates, along with fertility rates, are one of the immediate determinants of the population growth of a country. The relationship between mortality and fertility rates controls the population growth in a dynamic equilibrium and directs the demographic structure of the population as well as the demographic transition.

There are several pieces of evidence to show that there has been a rapid reduction in child mortality rates over the course of modernisation characterised by a decline in poverty and improvements in health. The dynamics of demographic transition primarily depend upon the birth rates and death rates. Generally, it is observed that in most developing countries high fertility rates are coupled with high mortality rates. Similarly, in developed countries, there exists low fertility rates and low mortality rates. Standard demographic transition theory posits that mortality rates for infants and children drive fertility rates, intermediated through cultural, social and psychological processes. Those social processes have the effect of keeping population size stable. Under conditions of high mortality, high fertility rates are required to keep the population from shrinking. When there is a transition from high to low mortality rates, fertility rates decrease.

Many studies also suggest that the relationship between fertility and mortality is bi-directional. One side of the argument is that as the number of children per woman increases the time and resources that are shared between the children get reduced and this could increase the probability of a child dying at an early age. The other side of the argument is that as the mortality risks are high, given the parental expectation of the ideal number of births, higher would be the fertility rates to cope with the expected levels of births. Therefore, when the mortality risks are high the fertility rates also tend to be higher. First, parental expectations of child loss might be expressed in terms of insurance or hoarding behaviour causing fertility to be higher than if survival were assured. In terms of the life cycle theory, the parents face a trade-off between the number of children and the quality of care given to each child. The rise in childbirth reduces available time and energy to invest in each individual child, increasing their likelihood of dying. At the same time, with post-natal investment in offspring, the death of an existing offspring releases time

and energy for the mother to invest in an additional child, a placement offspring. Initially, fertility and child mortality around the world were so high that on average every woman lost 1 of her children before they could celebrate their 5th birthday. With the decline of child mortality and fertility, this became very rare; in 2015 Swedish women lost on average 0.006 children before they were 5 years old. It is known that one in every two children dies and if the expected level of children that they wish to have is three, then their total number of births would naturally be six.

Similarly strong relationship exists between overall economic development and the levels of death rates. Economic development generally depicted by the gross domestic income per capita is also the best single indicator of the standard of living, As GDP per capita is weighted by the population, economic development influences mortality rates. Also, studies have observed that the relationship between income per head and levels of mortality is convex, that is a small gain in income per head in low-income countries tends to produce a decline in the mortality rates and the effect tends to flatten out as the income per head increases. It is also observed that only in situations where a country shares their health technology to control death rates, that mortality become significantly disassociated from economic levels. For example in Ceylon, Mauritius and British Guiana, low-cost public health services provided during the post-war era have resulted in a significant decrease in the death rates despite any significant increase in the levels of living.

Among all the factors associated with child mortality, female literacy has been the single most important factor in reducing child mortality. There is an inverse relationship between the levels of female literacy and infant mortality rates. Shetty and Shetty (2017) argue that female literacy is an empowerment as well as an emancipator; literate females enjoy greater autonomy and are more involved in decision-making. Literate women are able to adopt a healthier lifestyle and this has a great positive impact on mother's as well as child's health, thereby reducing child mortality rates as well as maternal mortality rates. The benefits of education for women are many and varied; one prime benefit is healthier children which again reduces the risk of child death. Education among females improves nutrition intake and personal hygiene, which has a significant impact on their own health as well as the health of their offspring. Short birth intervals and unintended pregnancies pose serious risks to the mother and her infants by causing a high risk of pregnancy-related complications and self-induced abortions. Therefore, education among females improves awareness of such problems and the knowledge of immunisation,

mitigating child mortality risks. Countries which have progressed more on the female literacy front reduce infant mortality faster. States and countries which have a higher percentage of literate women perform better on developmental and social indices, Women's education is also often cited as one of the most valuable tools to reduce poverty.

Figure 1 shows the various causal links that exist between a woman's education, child mortality and economic development (Browne and Barrett, 1991). The links shown are primarily health-related know-how.

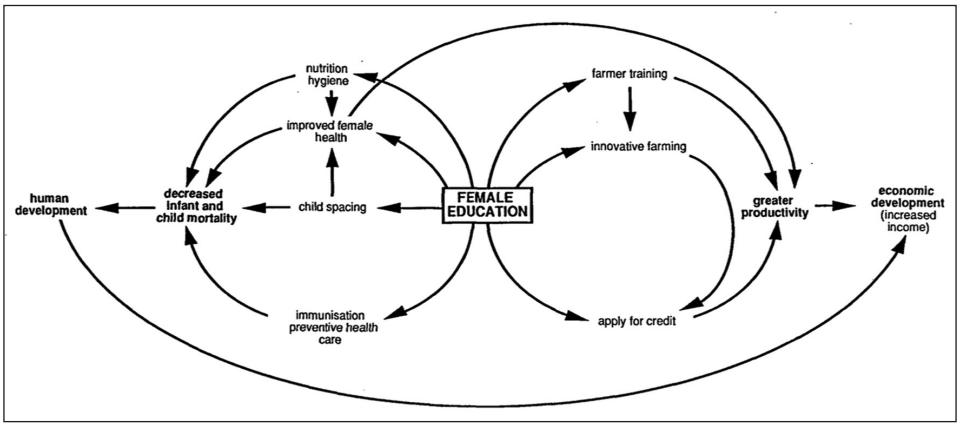

Figure 1: Causation Structure of Female Education on Child Mortality

Source: Browne and Barrett (1991).

The objective of this paper is to analyse the impact of the major factors that influence child mortality rates. Specifically, this paper estimates the effects of economic development, inequality, fertility, female literacy and female labour force participation on child mortality rates across countries and over time. The panel data used in this paper for 85 countries over the period 2000-2020 is derived from various sources including the World Development Reports and population databases. In the empirical, the panel fixed effect instrumental variable method is used.

2. Review of Literature

In the 1970s, Preston (1975) estimated the relative contribution of economic factors to increases in life expectancy, the opposite of mortality, exploring the cross-sectional relationships between national life expectancies and income per capita

during three different decades of the 20th century – 1900s, 1930s and 1960s. The study plots logistic curves that specify the relation between life expectancy at birth and national income per head. The curves thus estimated produced upward-sloping concave curves, shifting upward for each cross-section that was estimated. Also, the simple correlation between life expectancy and the logarithm of income per head was 0.885 in the 1930s and 0.880 in the 1960s. At higher income levels the shift is unmistakable; it amounts to some 10-12 years of life expectancy at incomes between $100 and $500 and progressively less thereafter due to the concavity in their relationship. A comparison of the curves horizontally and vertically suggested that, in order to attain a particular value of life expectancy between 40 and 60, a range that includes a large majority of the current world population, a nation required an income level of approximately 2-6 times higher in the 1930s than in the 1960s.

Hobcraft, Mcdonald and Rutstein (1984) analyse five socio-economic correlates of infant and child mortality using the World Fertility Survey for 28 developing countries. The study considers variations in mortality for the first five years of children for each of the following variables: mother's education, mother's work status, husband's occupation, husband's education and place of residence. A multivariate approach to assess the relative importance of each of the five variables reveals that the mortality differences are most strongly associated with the mother's education and occupation and the differences increase with age through the first five years of life of the child. The mother's education is strongly associated with mortality during the first five years of life particularly in Asian countries. Child mortality in a few African countries is relatively strongly associated with the husband's occupation and education. The mortality differences between the first and fourth birthdays of children are often appallingly large because of socioeconomic differences.

Torre and Myrskyla (2014) consider age-specific mortality rates and life expectancy as response variables, for the age groups of 1-14, 15-49, 50-64 and 65-89, to estimate the effects of GDP per capita and income inequality using a panel data for the period 1975-2006. The study observes that the GDP per head and unobserved period and country factors, income inequality, measured by the Gini index, are strongly and positively associated with male and female mortality up to age 15. For women the association vanishes at older ages, but for men persists up to age 50. These findings suggest that policies decreasing income inequality may improve the health of children and young middle-aged men.

Gerdtham and Johannesson (2004) examine whether mortality is related to individual income, mean community income and inequality in community income, controlling for initial health status and personal characteristics. The regression analysis is based on data from over 40000 individuals who were followed up for 1-17 years. The dependent variable in the regression analysis is the survival time in years and the survival status at the end of the follow-up period. To estimate the effect of income and other covariates on mortality risk, a Cox proportional hazard model is estimated. The estimates of the Cox model show that individual income is highly significant with a negative sign, implying that the mortality risk decreases with higher income. Further, the study suggests that the relationship is highly nonlinear with a decreasing effect of income at higher income levels. The other relationships are not statistically significant.

Miller and Paxson (2006) explore the relationship between relative income and mortality in the US using semi-aggregated data on mortality rates of people categorised by age, race, gender and place of residence for 1980 and 1990. The study develops an empirical model in which individual health is a function of own income and the incomes of those who live in the same geographical area. The study observes no significant relation of mortality with having relatively wealthy neighbours, and holding own income fixed. However, for working-aged black males having relatively wealthy neighbours is associated with lower mortality. For example, among younger (aged 25–64) black men, an increase in income of others is estimated to have a beneficial effect on mortality that is 40% as large as an equivalent increase in income.

Haines (1998) uses an instrumental variable technique to analyse the bi-directional relationship between child mortality and fertility rates. The instrument, the proportion of death, is regressed on fertility rates to obtain the fitted values, which are then regressed with the original first-stage regression equation. The estimated coefficient is a good predictor of the replacement or the hoarding effect.

Saurabh, Sarkar and Pandey (2013) investigate the relationship of male and female literacy rates with crude birth rates and infant mortality rates in the states and union territories of India. The estimated regression coefficients show that CBR is significantly inversely related to the female literacy rate, while the male literacy rate is not significantly related to CBR. The IMR of the states is also inversely related to their female literacy rate, whereas the male literacy rate is not significant. As educated women are known to make informed reproductive and healthcare

decisions, female literacy is highly important for both population stabilisation and better infant health care which is reflected in lower birth rates and infant mortality rates respectively.

Westoff and Bankole (2001) argue that as one of the major proximate determinants of fertility, contraceptive prevalence is expected to be strongly negatively associated with fertility. To analyse the differences in the relationship between contraceptive usage and fertility rates in sub-Saharan Africa and other regions, they consider three measures of fertility: births in the last 12 months, proportion currently pregnant and total fertility rate. The study finds a correlation of -0.368 between the total fertility rate and contraceptive usage for sub-Saharan Africa, however, for other regions it is -0.807. They advance various hypotheses to account for this anomaly: African women use more traditional methods with higher failure rates; contraception is used more for spacing than for limiting births implying more discontinuation; total fertility rate may be insensitive to more recent rapid increases in contraceptive prevalence. The study concludes that the populations in sub-Saharan Africa are clearly at the beginning of their fertility transition and can be compared with Asian and Latin American populations at much more advanced stages of the transition. By comparing the correlation for the same regions that are surveyed more than once, the study discerns a clear trend in sub-Saharan Africa toward a higher correlation over time.

The World Health Organisation (2015) reports that around 25-40% of maternal deaths could be eliminated if unplanned pregnancies are prevented. When the use of contraceptives is low in a country, the total fertility rate – the average number of children per woman during her lifetime – is often high. The contraceptive prevalence rates (CPR) in countries with a high MMR and a low proportion of skilled care at birth are usually low. The total fertility rate (TFR) in countries with a low CPR is usually high. Most of the countries with a high total fertility rate often have limited choice of contraceptive methods.

3. Data and Methodology

This paper uses cross-country panel data for 85 countries for the period 2000-2020 from the World Bank database to analyse the determinants of the mortality rates under the age of five. The child mortality rate is expressed as a function of fertility rate, per capita income, income inequality and female literacy and labour force participation rates. However, as there is a bi-directional causality between

fertility rates and child mortality rates, the contraceptive prevalence rate enters as an instrumental variable in the method of estimation. The dependent variable, under-five mortality rate is the number of children that died before reaching the age of five per 1000 children. The fertility rate is the number of children that would be born to a woman if she were to live to the end of her childbearing years and bear children in accordance with age-specific fertility rates of specified years. The female literacy rate is the percentage of females aged 15 and above who can both read and write. The GDP per capita is the per-person value of the goods and services produced within a country during a given year (weighted by the population of the country at constant 2010 USD). The Gini index measures the area between the Lorenz curve and the line of absolute equality, expressed as a percentage of the maximum area under the line. Female labour force participation shows the extent to which women are active in the labour force. The contraceptive prevalence rate, included as an instrumental variable, is the percentage of women aged 15-49 years who are married or in a union and who are practising or whose sexual partners are practising any form of contraception.

The major advantage of a panel data set over a cross-section is that it allows greater flexibility in modelling differences in behaviour across individuals. The heterogeneity or individual effects λ_i may be observed or unobserved. The empirical analysis begins with the pooled OLS method:

$$y_{it} = \alpha + \beta x_{it} + \lambda_i + u_{it} \tag{1}$$

In the panel data context, the pooled regression model is the population-averaged model as, under the classical model assumptions, any latent heterogeneity is averaged out.

If λ_i are taken to be constant over time, then first differencing is just one of the many ways to eliminate the individual effect λ_i. An alternative method is the fixed effects or within transformation. For each i, averaging equation (1) over time yields:

$$\overline{y}_i = \beta \overline{x}_i + \overline{u}_i \tag{2}$$

where $\overline{y} = T^{-1}\Sigma_{t=1}^{T} y_{it}$. Subtracting equation (2) from equation (1) eliminates the unobserved individual heterogeneity λ_i which is fixed over time. The resulting time-demeaned transformed equation is:

$$(y_{it} - \overline{y}_i) = \beta(x_{it} - \overline{x}_i) + (u_{it} - \overline{u}_i) \tag{3}$$

As the unobserved individual effect λ_i has disappeared, equation (3) can be estimated by the pooled OLS on the time-demeaned variable as fixed effects

estimator or the within estimator. However, when λ_i is correlated with x_i, the least squares estimator of β is biased and inconsistent as a consequence of an omitted variable. If there exists some observable fixed effects, $\alpha_i=z_i\alpha$, the fixed effects model approach takes α_i to be a group-specific constant term in the regression model. Then, the model:

$$y_{it} = \alpha_i + \beta x_{it} + u_{it} \tag{4}$$

specifies an estimable conditional mean.

If the unobserved individual heterogeneity λ_i, however formulated, is uncorrelated with x_i, then the model can be formulated as a linear regression model with a compound disturbance term:

$$y_{it} = \beta x_{it} + E[z_i\alpha - E(z_i\alpha)] + \varepsilon_{it} \tag{5}$$

$$\text{yit} = \beta x_{it} + \beta + u_i + \varepsilon_{it} \tag{6}$$

This random effects approach specifies that u_i is a group-specific random element. The crucial distinction between fixed and random effects is whether the unobserved individual effect embodies elements that are correlated with the regressors in the model, not whether these effects are stochastic or not. The random effects model, as a linear regression model with a compound disturbance, may be consistently, although inefficiently, estimated by least squares.

However, when there is an endogenous regressor in the model, the covariance between the endogenous variable and error term is not zero, hence the OLS-type estimates tend to overestimate the parameters and therefore the estimates are biased and inconsistent. The problem of endogeneity arises when there is bi-directional causality between the dependent and the independent variable as the error u is directly affecting the regressors x and therefore indirectly affecting the dependent variable y. The structural equations of the model can be specified as:

$$x_{it} = \beta_0 + \beta_1 y_{it} + \beta_2 z_{ti} + u_{it} \tag{7}$$

$$y_{it} = \alpha_0 + \alpha_1 x_{it} + e_{it} \tag{8}$$

Inserting equation (8) in equation (7) implies that x_{it} is a function of e_{it} and hence $cov(x_{it}|e_{it}) \neq 0$. Similarly, plugging equation (7) in equation (8) shows that y_{it} is a function of u_{it}, and hence cov $(y_{it}|u_{it}) \neq 0$.

Therefore, the correlation between the endogenous variable x and error term u is to be removed using an instrumental variable (IV) estimation method. The IV is a variable z that has the property that changes in z are associated with changes in x

but do not lead to changes in y and are not correlated with u, thus introducing only exogenous variation in x. The IV estimation considers the covariance between the instrument z and the dependent variable y:

$$\operatorname{cov}(z_i, y_i) = \operatorname{cov}[z_i, (\beta_0 + X'\hat{\beta} + u_i)] = \operatorname{cov}(z_i, \beta_0) + \hat{\beta}\operatorname{cov}(z_i, x_i) + \operatorname{cov}(z_i, u_i) \tag{9}$$

By the assumption of the instrumental variable method, the IV is uncorrelated with the error term:

$$\operatorname{cov}(z_i, u_i) = 0 \Rightarrow \operatorname{cov}(z_i, y_i) \Rightarrow \hat{\beta}\operatorname{cov}(z_i, x_i) \Rightarrow \hat{\beta} = \frac{\operatorname{cov}(z_i, y_i)}{\operatorname{cov}(z_i, x_i)} \tag{10}$$

The IV estimation is also a two-stage least squares estimation. Specifying the model as structural equations:

$$y = \beta_1 x_1 + \beta_2 x_2 + u \tag{11}$$

where y is the vector of the dependent variable, x_1 is the matrix endogenous variable and x_2 is a matrix of exogenous variable. The structural equations model involves a combined set of $x = [x_1, x_2]$ of both endogenous and exogenous independent variables. The set of instruments is specified for only exogenous variables as $z = [z_1, x_2]$ where z_1 is an instrument for x_1 and x_2 is an instrument for itself. The 2SLS method replaces the endogenous variable with the predicted values of this endogenous variable when regressed on the instrument chosen. First, the first stage equation with only exogenous regressors is estimated:

$$x_1 = \gamma_1 z + \gamma_2 x_2 + \varepsilon \tag{12}$$

Then, the fitted values of $\hat{x}_1$ are substituted in the structural equation:

$$y = \hat{\beta}_1 \hat{x}_1 + \hat{\beta}_2 x_2 + u \tag{13}$$

The fixed effects model allows the unobserved individual effects to be correlated with the included variables and the differences between units are parametric shifts of the regression function. In the random effects model the individual specific constant terms are randomly distributed across cross-sectional units, thereby greatly reducing the number of parameters to be estimated. An inevitable question is which should be used. From a purely practical standpoint, the dummy variable approach is costly in terms of degrees of freedom lost. On the other hand, the fixed effects approach is superior as there is little justification for treating the individual effects as uncorrelated with the other regressors, as is assumed in the random effects model. The random effects treatment, therefore, may suffer from inconsistency due to this correlation between the included variables and the random effect.

The specification test devised by Hausman (1978) is used to test for orthogonality of the common effects and the regressors. The test is based on the idea that under the hypothesis of no correlation, OLS, fixed effects and random effects estimators are consistent, but OLS is inefficient, whereas under the alternative, the fixed effect is consistent, but the random effect is not. Therefore, under the null hypothesis, the two estimates should not differ systematically, and a test can be based on the difference. The other essential ingredient for the test is the covariance matrix of the difference vector $[b-\hat{\beta}]$:

$$\text{var}[b-\hat{\beta}] = \text{var}[b] + \text{var}[\hat{\beta}] - \text{cov}[b,\hat{\beta}] - \text{cov}[\hat{\beta},b] \tag{14}$$

Hausman's essential result is that the covariance of an efficient estimator with its difference from an inefficient estimator is zero, which implies that:

$$\text{cov}[(b-\hat{\beta}),\hat{\beta}] = \text{cov}[b,\hat{\beta}) - \text{var}[\hat{\beta}] = 0 \Rightarrow Cov[b,\hat{\beta}] = \text{var}[\hat{\beta}] \tag{15}$$

Inserting equation (15) in equation (14) produces the covariance matrix for the Hausman test:

$$\text{var}[b-\hat{\beta}] = \text{var}[b] - \text{var}[\hat{\beta}] = \psi \tag{16}$$

The test statistic is thus:

$$H = (b_{FE} - \hat{\beta}_{RE})'(\psi_{FE} - \psi_{RE})^{-1}(b_{FE} - \hat{\beta}_{RE}) \tag{17}$$

The chi-squared test is based on the Wald criterion:

$$W = \chi^2(k-1) = [b-\hat{\beta}]\hat{\psi}^{-1}[b-\hat{\beta}] \tag{18}$$

For the fixed effects model, the estimated covariance matrix ψ is the slope estimator and for the random effects model, the estimated covariance matrix ψ excludes the constant term. The null hypothesis and alternative hypothesis for the Hausman test are specified as H_0: $b = \hat{\beta}$ and H_1: $b \neq \hat{\beta}$. When the computed value of the testing statistic is greater than the critical value, the null hypothesis of the random effects model is rejected and the preferred specification for the data is the fixed effects model.

4. Empirical Analysis

The estimating empirical panel model specifies an endogenous relationship between child mortality rate and fertility rate, along with other covariates like per capita income, Gini index, female literacy rate and female labour force participation rate.

The theoretical argument for the endogeneity of the variables is that higher the fertility rates, the parents will have less time and resources to devote to the newborn child hence causing the death of that child at an early age. In the other direction, the higher the mortality rate the parents would give birth to more children to meet their required number of births. When there is bidirectional causality between two variables, then the error terms will be correlated with the current endogenous variable, the fertility rate. Therefore, the instrumental variable method is used when there is bidirectional causality between the two variables. The structural equations are given as:

$$lnCMR_{it} = \beta_0 + \beta_1 lnGDPPC_{it} + \beta_2 lnGI_{it} + \beta_3 LR_{it} + \beta_4 FR_{it} + \beta_5 FLPR_{it} + \lambda_i + u_{it} \quad (19)$$

$$FR_{it} = \alpha_0 + \alpha_1 lnMR_{it} + \delta_i + \varepsilon_{it} \quad (20)$$

where λ_i represents the individual heterogeneity. Plugging equation (20) into equation (19) implies that the mortality rate is a function of ε_{it}. Hence, the $\text{cov}(lnMR_{it} \mid \varepsilon_{it}) \neq 0$. Similarly, plugging equation (19) into equation (20) implies that the fertility rate is a function of u_{it}. Hence, the $\text{cov}(FR_{it} \mid u_{it}) \neq 0$. Therefore, an instrumental variable which is uncorrelated with the error term u_{it} and associated with the endogenous covariate is introduced. The instrument that is used for the fertility rate which is the endogenous variable is the contraceptive prevalence rate (CPR). There are several studies that show a strong negative relationship between the contraceptive prevalence rate and the fertility rate. The condition that is to be satisfied by CPR as the instrumental variable is:

$E(CPR_{it}, u_{it}) = 0$, the CPR is uncorrelated with the error term.

$E(CPR_{it}, FR_{it}) \neq 0$, the CPR is correlated with the endogenous variable, the fertility rate.

The CPR is not a direct cause of dependent variable MR, the $Cov\ (MR_{it}, CPR_{it} \mid FR_{it}) = 0$.

In other words, the CPR is not included in the structural equation.

Comparing the estimated effects with and without instruments, the degree of upward and downward biases due to the non-zero covariance that exists between the time-invariant unobserved heterogeneity and the covariates can be ascertained.

Table 1 presents the descriptive statistics of the variables used in the IV analysis of the determinants of child mortality for panel data of 85 countries for the period 2000-2020. The mean child mortality rate is 68.1 with a standard deviation of

49.61. The high standard deviation signifies the significance of the time-variant and invariant as well as observed and unobserved heterogeneity within and between the countries. For example, the differences in the national income between countries can explain the high standard deviation in the child mortality rates. The high variation in the national income is explained by the standard deviations in the GDP per capita, which is US$ 7880.46, with a mean of US$ 3137.42. Similarly, female literacy also has a high standard deviation of 27.14 with a mean of 66.26. The mean of the Gini index that measures income inequality is 38 with a standard deviation of 9.24. The labour force participation rate has a mean of 40.65 and a standard deviation of 9.051.

Table 1: Descriptive Statistics of Variables

Variable	*Mean*	*Standard deviation*
Child mortality rate (CMR)	68.61	49.61
Fertility rate (FR)	3.88	1.63
GDP per capita (GDPPC)	3137.42	7880.46
Gini index (GI)	38	9.24
Female literacy rate (FLPR)	66.26	27.14
Female labour participation rate (FLR)	40.65	9.051

Table 2: Panel IV Estimates of Child Mortality Rate

Variable	*Pooled OLS*	*Fixed effects*		*Random effects*
		Not instrumented	*Instrumented*	
Fertility rate	0.224* (0.04)	0.097** (0.05)	0.318* (0.09)	0.273* (0.06)
Ln(GDP per capita)	-0.354* (0.03)	-0.353* (0.29)	-0.297* (0.04)	-0.308* (0.03)
Ln(Gini index)	0.717* (0.15)	0.102 (0.17)	0.141 (0.19)	0.360* (0.14)
Female literacy rate	-0.006* (0.002)	-0.008** (0.003)	-0.007* (0.003)	-0.005*** (0.002)
Female labour force participation rate	-0.002 (0.003)	0.0005 (0.12)	0.009 (0.14)	0.003 (0.004)
Constant	5.120* (0.50)	6.32* (1.06)	4.37* (1.41)	3.90* (0.77)
R-square (within)	-	0.82	0.78	0.79
R- square (between)	-	0.83	0.83	0.85
R-square	0.86	0.84	0.83	0.85
Rho-value	-	0.94	0.93	0.91

Note: Standard errors in parenthesis. *, **, *** Significant at 1, 5. 10% levels.

The null hypothesis of Hausman's test is that there is the random effect or differences between the models are not systematic whereas the alternative hypothesis states that there are no differences and the presence of fixed effects. The chi-square value for the Hausman's test is highly significant confirming that the difference in the coefficients of the fixed and random effects model are significant, which suggests that there exists a strong relation between time-invariant unobserved individual heterogeneity and the covariates in the model. Hence, the null hypothesis that there is a random effect is rejected and hence the fixed effects model is the appropriate model. Thus, the fixed effects model is considered to be an appropriate model to obtain unbiased and consistent estimates. The fixed effect model controls the country-wise heterogeneity due to differences in economic, social and demographic factors across regions. The country-wise time-invariant unobserved heterogeneity can be the lifestyles that a particular country may follow, levels of immunity to diseases, gender bias, general hygiene, knowledge of implementing health-related techniques into child rearing etc.

The panel IV estimates of child mortality are presented in Table 2. In the fixed effects model, the within R-square value for the model is 0.78 showing about 78% variation in child mortality rates in each of the countries is mainly explained by their per capita income, fertility rates and female literacy rates through time. Similarly, the between-group R-square states that 83% of the variation in child mortality rates between countries is explained by the covariates that enter into the model. The rho value of 0.94 shows that about 94% of the variance in child mortality is due to differences across panels. This statistic indicates that there exists a wide inequality across the countries around the world showing polar opposites; on one hand, there are countries which exhibit a very promising situation of child mortality, and on the other hand, there are nations with alarming situations with regard to child mortality.

The panel estimates show that the log of GDP per capita and female literacy rate are significantly negatively related to the log of the mortality rate of the countries around the world. This is in line with the findings of Torre and Myrskyla (2014). Per capita income shows the general economic development in the countries which could significantly reduce the levels of child mortality rates. A one-unit increase in per capita GDP leads to a decrease of 0.29 units of log of child mortality rate. An increase in per capita GDP induces the ability of countries to obtain higher standards of living thus contributing to declining child mortality. Also, a one-unit

increase in female literacy rate leads to a 0.008 unit decrease in child mortality rates in the countries. Literate mothers are able to incorporate healthy lifestyles and hence female literacy has a significant impact on the child's health. This finding is in line with the findings of Saurabh, Sarkar and Pandey (2013).

The estimated effect of fertility rate is highly significant in all specifications and is positively associated with child mortality rates. A unit increase in total fertility rate induces an increase of 0.318 units in child mortality rates. The fertility rate is instrumented with the contraceptive prevalence rate to obtain an unbiased estimate of the impact of the fertility rate on the child mortality rate. In order to assess the strength of the instrumental variable and the identification of the structural equation, three tests are considered: Anderson-Canon Lagrange Multiplier, Wald F and Sargan-Hansen tests for identification. The test results presented in Table 3 confirm that the equation is not under-identified since the computed Wald F-statistic is greater than the critical value. Therefore, the instrument contraceptive prevalence rate is a strong instrument for the endogenous regressor fertility rate.

Table 3: Identification Test Statistics

Identification test	Test statistic	Test statistic value
Under identification	A-C LM statistic	19.918 (0.00)
Weak identification	Wald F statistic	24.629
Over identification	Sargan-Hansen statistic	21.236 (0.00)

Note: p-values in parentheses.

Even though in the fixed effects model the Gini coefficient is not significant enough to explain the variation in child mortality, the estimated coefficient shows a positive relation between the two, similar to Torre and Myrskyla (2014) which is statistically significant in their study. This means that as income inequality rises child mortality rate also increases. However, the literature is not able to identify the exact dynamics through which inequality affects child death rates. Similarly, the female labour force participation rate has no statistically significant impact on child mortality rates.

5. Conclusion

This paper examines the effects of per capita income, income inequality, female literacy rate, fertility rates and female labour force participation rate on child mortality rate in a panel of 85 countries for the period 2000-2020. The study uses

a fixed effects instrumental variable method with the fertility rate as an endogenous variable in the model. The empirical estimates show a significant negative effect of per capita income and female literacy rate and a strong positive impact of fertility rate on child mortality in the panel countries. However, the analysis also finds no significant role for income inequality and female labour force participation in controlling child mortality. Thus, in countries where there is persistent child mortality, the policies should focus on bringing down the fertility rates through education, especially female literacy and more so on the usage of contraceptive usage. The strong positive relationship between overall economic development and the levels of death rates strengthens the argument that economic development is the best contraceptive. Increasing income per capita also encompasses other components of living standards, such as health and lifestyle changes. Undoubtedly, controlling child mortality rates is an advantage to the population with considerably less pressure on resource use and an even greater advantage to the growth of the economy.

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