Socio-economic Determinants of Infant Mortality Rate

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Abstract

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Abstract

This study investigates the socio-economic determinants of infant mortality rates using panel data from the World Bank's World Development Indicators (WDI) for over 100 countries spanning multiple decades. Given the potential endogeneity in the fertility-mortality relationship, a two-stage least squares (G2SLS) instrumental variable (IV) approach is employed, using the second lag of fertility as an instrument. This strategy accounts for reverse causality and omitted variable bias, ensuring more robust estimates of the causal effect of fertility on infant mortality. A fixed-effects regression model is implemented to analyze the dynamic effects of fertility rate, real GDP per capita, urbanization, and economic growth on infant mortality, incorporating interaction terms to examine how economic development moderates the impact of fertility. The results confirm that higher fertility rates are associated with increased infant mortality, but this effect diminishes in wealthier economies, highlighting the role of economic development in mitigating child mortality risks. Additionally, urbanization and short-term economic growth exhibit significant negative associations with infant mortality, underscoring the importance of healthcare access and economic stability in improving child health outcomes.

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1 Introduction

Mortality rate, defined as the number of deaths among children under one year per 1,000 live births, serves as a widely recognized indicator of a nation's health, socio-economic progress, and development. It reflects the effectiveness of healthcare systems and broader socio-economic policies. The United Nations' Sustainable Development Goals (SDGs) prioritize the reduction of child mortality, setting ambitious targets that emphasize the need to understand its determinants and design effective interventions. While global child mortality has declined significantly over recent decades, substantial disparities persist across countries. Figure 1 presents the year-on-year average mortality rate by income classification. Mortality rates remain significantly higher in low-income countries compared to wealthier ones. The data, sourced from the World Bank, follows the institution's income classification system, highlighting stark inequalities in child survival. These disparities underscore the complex socio-economic factors influencing infant health and raise critical questions about the policies needed to address them effectively.

Theoretical and empirical evidence highlights the crucial role of economic development in shaping mortality rate. Gross domestic product (GDP) per capita, often used as a proxy for economic prosperity, consistently demonstrates a negative relationship with mortality. Preston (1975) seminal work on the relationship between mortality rate and economic development reveals that even modest increases in income per capita can lead to significant reductions in mortality in low-income countries. However, the impact of income levels tends to plateau in wealthier nations, suggesting that the relationship between economic development and mortality rate is not linear. Similarly, Bongaarts (2017) finds that urbanization and access to quality healthcare, which often accompany economic growth, contribute to lower mortality rates by improving sanitation and healthcare infrastructure. However, Garenne (2010) observes that rapid and unplanned urbanization can also lead to overcrowding and inadequate healthcare services, potentially offset by these benefits.

Fertility rates play a similarly pivotal role in shaping infant mortality. Hobcraft, McDonald, and Rutstein (1984) argues that high fertility rates, prevalent in many low-income countries, place significant pressure on household resources and healthcare systems, increasing the risks for both mothers and children. However, this relationship is not uniform across all contexts. In wealthier nations with stronger healthcare systems and better access to family planning, the negative effects of high fertility on infant mortality are less pronounced. Haines (1998) further highlight the bidirectional nature of this relationship, noting that high child mortality often incentivize higher fertility as a replacement strategy, complicating causal analysis.

Beyond economic growth and fertility, other socio-economic factors significantly shape mortality rate trends. Income inequality, often measured by the Gini index, is a crucial determinant, high inequality can restrict access to quality healthcare, education, and nutrition, essential components for reducing infant mortality. Deaton (2003) argue that countries with greater income inequality often experience poorer health outcomes, even when accounting for average income levels, as wealth concentration limits healthcare accessibility for lower-income population.

Labor force participation among women significantly influences infant mortality through multiple pathways. Heath and Jayachandran (2017) finds that when women join the workforce, they gain economic and social empowerment, which helps them make better healthcare decisions, improve child nutrition, and invest in early childhood development. However, Nandi et al. (2016) emphasizes that increasing female labor force participation alone is not enough, as the absence of supportive policies, such as paid maternity leave and accessible childcare, can reduce these benefits. Their study showed that inadequate maternity leave policies often force mothers to return to work too soon after childbirth, limiting time for breastfeeding, postnatal care, and essential maternal-infant bonding, all of which are critical for lowering infant mortality. They further find that in low and middle income countries, longer paid maternity leave is linked to significant reductions in infant mortality rates, suggesting that labor policies play a key role in improving child health outcomes. While greater female workforce participation can enhance household well-being, the lack of institutional support can undermine these gains. This highlights the need for policies that allow women to work while ensuring the health and well-being of both mothers and children.

Despite extensive research on the socio-economic determinants of child mortality, key gaps remain. Existing studies have established that economic growth, urbanization, and fertility rates influence mortality, but the way these factors interact remains less understood. In particular, the extent to which economic conditions shape the impact of fertility on infant mortality is unclear. High fertility may contribute more to mortality in low-income settings due to resource constraints, while in wealthier nations, stronger healthcare systems may mitigate its effects. This study addresses these gaps by analyzing the socio-economic determinants of infant mortality using a dataset covering over 100 countries. It examines how fertility rates, GDP per capita, and their interaction shape child survival outcomes. Additionally, it incorporates urbanization and short-term economic growth to provide a comprehensive understanding of the drivers of infant mortality.

A key contribution of this research is the inclusion of an interaction term between fertility rates and GDP per capita, which allows for a more nuanced analysis of how economic development moderates the relationship between fertility and mortality. In low-income countries,

where health care systems and resources are more constrained, high fertility rates may contribute more significantly to increased mortality. In contrast, in high-income countries, with better healthcare and social services, the association between fertility and mortality may be weaker. By accounting for these differential effects, this study extends existing research by providing a context-sensitive analysis of how economic and demographic factors interact to shape child mortality.

To address potential endogeneity in the fertility-mortality relationship, an instrumental variable (IV) approach is employed, using the second lag of fertility as an instrument. The rationale for this choice is that past fertility decisions influence current fertility rates while remaining exogenous to contemporaneous shocks to infant mortality. This approach strengthens the causal interpretation of the findings, offering more robust estimates compared to traditional fixed-effects models that do not correct for endogeneity.

This paper is structured as follows. Section 2 reviews the theoretical framework and empirical literature on the socio-economic determinants of infant mortality. Section 3 and 4 describes the methodology and data, detailing the fixed-effects regression model and instrumental variable strategy. Section 5 presents the results, highlighting the role of fertility, economic development, and other socio-economic factors. Section 6 discusses the implications of the findings for policy and future research. Finally, Section 7 concludes with key takeaways and recommendations for targeted interventions to reduce infant mortality globally.

2 Literature Review

A vast body of research has explored the factors influencing infant mortality, revealing that it is shaped by a complex interplay of economic conditions, healthcare access, fertility patterns, and broader socio-demographic characteristics. This review summarizes findings from key studies examining the socioeconomic determinants of mortality rate, with a particular emphasis on fertility rates, GDP, income inequality, urbanization, and female labor force participation. By evaluating both cross-country and country-specific studies, this review aims to identify the key drivers of mortality rate and highlight the mechanisms through which these factors operate. Moreover, it considers the methodological approaches used in prior research, particularly the use of IV estimation to improve causal inference. Understanding these relationships is essential for developing effective policy interventions aimed at reducing infant mortality in developing regions.

A central theme in child mortality research is the extent to which economic development influences child survival. Preston (1975) provides foundational evidence showing that

income per capita correlates inversely with child mortality, with wealthier countries experiencing lower mortality rates. Cutler, Deaton, and Lleras-Muney (2006) expand on this finding, demonstrating that economic growth leads to rapid reductions in child mortality in low-income countries, but its impact diminishes as income levels rise. This suggests that while economic progress improves healthcare, nutrition, and living standards, beyond a certain point, additional income does not necessarily translate into better child survival outcomes. Some researchers argue that GDP per capita alone is an imperfect measure, as it fails to capture how wealth is distributed within a society. Dědeček and Dudzich (2021) argue that GDP per capita, even when adjusted for purchasing power parity, has significant drawbacks as an indicator of economic development and well-being, as it overlooks how wealth is distributed among the population. These perspectives underscore the necessity of considering income distribution metrics, such as the Gini coefficient, alongside GDP per capita to gain a more comprehensive understanding of economic prosperity.

Building on this, research increasingly shows that economic inequality rather than just absolute income levels—plays a crucial role in shaping child mortality outcomes. Deaton (2003) argues that even in high-income countries, greater inequality leads to worse health outcomes by limiting access to healthcare, education, and nutritional resources for marginalized populations. Schell et al. (2007) show that while national income strongly predicts child mortality, countries with high-income inequality experience slower reductions in child deaths. This aligns with the relative income hypothesis, which suggests that economic disparities, not just poverty, exacerbate health outcomes by limiting access to essential public services. However, the empirical evidence remains mixed. Pickett and Wilkinson (2015) demonstrate that government healthcare spending and social policies mediate the relationship between inequality and child mortality, suggesting that policy interventions can mitigate the effects of economic disparities on child survival.

Another widely studied determinant of child mortality is fertility rate, particularly in low-income settings where high birth rates are often associated with limited parental resources, poor maternal health, and increased risks of infant mortality. Hobcraft, McDonald, and Rutstein (1984) find fertility as a significant predictor of mortality rate, with high birth rates correlating with lower investments in child health and education. More recent studies reinforce this relationship. Bongaarts (2017) finds that declining fertility rates contribute to better maternal and infant health outcomes. However, the mechanisms underlying this relationship are complex and context dependent. Cleland and Jejeebhoy (2019) argue that in high-income settings with strong healthcare infrastructure, high fertility does not necessarily lead to higher child mortality, whereas in resource-constrained environments, it exacerbates risks by increasing maternal depletion, reducing birth spacing, and straining household re-

sources. LeGrand and Phillips (1996) support this view, showing that the negative effects of high fertility are more pronounced where healthcare access is limited. These findings emphasize the need for analyses that account for how economic and healthcare conditions shape the relationship between fertility and child mortality.

While the individual effects of fertility rates and economic development on mortality rate are well-documented, fewer studies have explicitly examined their interaction. Theoretically, the effect of fertility on child survival should depend on a country's economic context—high fertility rates may have more severe consequences for child survival in low-income settings than in wealthier nations with advanced healthcare systems. Becker (1960) first introduced the quantity-quality tradeoff theory, suggesting that in poorer households, high fertility reduces parental investments per child, leading to worse health and educational outcomes. In contrast, wealthier societies may be better able to mitigate the negative effects of high fertility through improved healthcare access and social safety nets. Empirical studies have provided some support for this hypothesis. Pampel Jr and Pillai (1986) found that the negative association between fertility and child mortality is significantly weaker in high-income countries, where better healthcare infrastructure and economic resources reduce child mortality risks. Similarly, Shandra, Shandra, and London (2011) demonstrates that high fertility is more strongly associated with infant survival rate in countries with lower GDP per capita, reinforcing the idea that economic development buffers the adverse effects of high fertility on child survival.

Incorporating an interaction term between fertility rate and GDP per capita in statistical models can provide a nuanced understanding of how these factors jointly influence infant mortality rates. This approach acknowledges that the impact of fertility on infant mortality is not uniform across different economic contexts. For instance, O'Hare et al. (2013) conducts a systematic review and meta-analysis, finding that in low-income countries, higher GDP per capita is associated with significant reductions in infant mortality rates, suggesting that economic development can mitigate the adverse effects of high fertility . Similarly, Ge et al. (2011) analyzed regional disparities in China and found that in areas with lower GDP per capita, increased hospital delivery rates were significantly associated with reduced neonatal mortality, highlighting the importance of healthcare interventions in economically constrained regions. While these regional studies offer valuable insights, comprehensive cross-country analyses incorporating this interaction remain limited. By explicitly modeling the interplay between fertility and economic development, this thesis aims to contribute to a more detailed understanding of how demographic and economic factors together shape infant mortality outcomes.

Urbanization has also emerged as an important but complex factor influencing infant

survival rate. The conventional view suggests that urbanization lowers child mortality by increasing access to healthcare, sanitation, and education. Garenne (2010) finds that infant mortality rates are generally lower in urban areas due to better healthcare facilities and higher literacy rates. However, the benefits of urbanization are not uniform—rapid, unplanned urban growth can lead to overcrowding, pollution, and the spread of infectious diseases, which may offset the positive effects. This suggests that while urbanization presents opportunities for reducing child mortality rate, its impact depends on the quality of urban planning and public health policies.

In addition, female labor force participation is one of the key factors that has received increasing attention in mortality rate research. Higher female labor force participation is often linked to better child health outcomes, as working mothers have greater financial autonomy and decision-making power over healthcare and nutrition. Kürkcu and Kandemir (2017) finds a significant negative relationship between women's workforce participation and child mortality, suggesting that economic empowerment of women leads to better infant survival rates. However, Heath and Jayachandran (2017) emphasize that this relationship is bidirectional. While financial stability improves healthcare access, increased workforce participation without adequate maternity leave or childcare support can reduce maternal care time, potentially increasing child mortality rates. The role of labor policies in moderating these effects remains an important area of research.

To improve causal inference in child mortality research, many studies employ instrumental variable (IV) approaches to address endogeneity concerns, particularly those arising from reverse causality and omitted variable bias. For example, Westoff and Bankole (2001) have used contraceptive prevalence as an instrument for fertility, this approach assumes that contraception primarily affects child mortality through fertility, an assumption that may not hold universally. Similarly, Almond and Mazumder (2011) utilizes access to prenatal care as an instrument, arguing that maternal healthcare directly influences fertility decisions while simultaneously improving infant survival rates. However, these instruments may suffer from potential direct effects on child health, making their exogeneity assumptions less robust in some contexts.

Given these concerns, this study employs the second lag of fertility rate ($\log FR_{it-2}$) as an instrumental variable. The choice of this instrument is motivated by the assumption that past fertility decisions influence current fertility behavior but are unlikely to be directly affected by contemporaneous shocks to infant mortality. Moreover, while the existing literature provides extensive insights into the socio-economic determinants of infant mortality rates, key gaps remain. Economic growth, healthcare access, and fertility reductions are well-documented contributors to improved child survival, yet their interaction is less studied. To

address this gap, this paper includes the interaction term ($\log FR_{it-2} \times \log Y_{it}$) to capture how economic conditions moderate the persistence of fertility, ensuring that the instrument varies with macroeconomic factors. This strategy strengthens the identification of causal effects by leveraging exogenous variation in fertility patterns that are plausibly unrelated to unobserved determinants of infant mortality.

Overall, the key contribution of this paper lies in its methodological approach, which combines instrumental variable estimation with a focus on the interaction between fertility and economic development. By employing a robust identification strategy, this study provides new empirical evidence on how macroeconomic conditions shape the fertility-mortality relationship, offering valuable insights for policy interventions aimed at reducing infant mortality globally.

3 Methodology

This study examines the causal relationship between fertility and infant mortality by incorporating key economic and demographic determinants. The model accounts for economic development, urbanization, and income growth to provide a comprehensive assessment of the factors influencing child mortality.

The empirical specification is as follows:

$$\begin{aligned}\Delta \log(IMR_{it}) = \beta_1 \log(FR_{it}) + \beta_2(\log(FR_{it}) \times \log(Y_{it})) + \beta_3 \log(Y_{it}) \\ + \beta_4 \Delta \log(Y_{it}) + \beta_5 \log(U_{it}) + \sum_t \gamma_t \text{Year}_t + \epsilon_{it}\end{aligned} \tag{1}$$

In this model, the dependent variable $\Delta \log(IMR_{it})$ represents the log difference in infant mortality, capturing percentage changes over time rather than absolute levels. This transformation allows the analysis to focus on dynamic changes in infant mortality rather than cross-country differences. The primary explanatory variable, $\log(FR_{it})$, measures the fertility rate, which is expected to have a direct effect on infant mortality through resource constraints, maternal health, and healthcare access. Economic development, represented by $\log(Y_{it})$, is included to capture the influence of income levels on child mortality, as higher incomes are typically associated with better healthcare and improved living conditions. The interaction term ($\log(FR_{it}) \times \log(Y_{it})$) is crucial for assessing whether economic development moderates the relationship between fertility and infant mortality. The inclusion of first-differenced GDP per capita, $\Delta \log(Y_{it})$, allows the model to capture short-term eco-

nomic fluctuations that may influence child mortality rates. Additionally, $\log(U_{it})$, which represents the urban population share, accounts for the role of urbanization in improving access to healthcare and sanitation, both of which contribute to reductions in infant mortality. The model also includes year fixed effects, $\sum_t \gamma_t \text{Year}_t$, to control for global trends and macroeconomic shocks that may affect multiple countries simultaneously. Finally, ϵ_{it} is the error term capturing unobserved influences on infant mortality.

By structuring the model in this way, the analysis effectively isolates the effects of fertility and economic development on infant mortality while accounting for broader demographic and economic shifts. The use of differencing ensures stationarity in the dependent variable, mitigating concerns of spurious correlations, and justifies the exclusion of country fixed effects, as differencing already removes time-invariant country-specific factors. This approach allows for a more precise estimation of the relationships between fertility, economic development, and child health outcomes across countries over time.

The key challenge in estimating the relationship between fertility and child survival rate is endogeneity, which arises due to several factors:

- **Reverse Causality:** Instead of fertility affecting child mortality, child mortality might be affecting fertility. For example, if more children are dying, parents might decide to have more children to replace those they lost. This creates a two-way relationship, making it hard to isolate the true effect of fertility on child mortality.
- **Omitted Variable Bias:** There could be unobserved factors that simultaneously influence both fertility and child mortality. For instance, healthcare infrastructure quality is a crucial determinant of infant mortality, but it is difficult to measure accurately across countries and over time due to data limitations. A country's healthcare system may also be shaped by cultural, institutional, or historical factors that are not explicitly included in the model.
- **Measurement Error:** Reporting inaccuracies in fertility rates and infant mortality data may distort the estimated relationship.

Hence, to obtain an unbiased estimate, an instrumental variable (IV) is required, one that is both relevant (strongly correlated with fertility) and exogenous (not directly affecting child mortality except through fertility). To address this, the second lag of fertility ($\log(FR_{it-2})$) is used as an instrument.

3.1 Relevance of the Instrument

The instruments must be correlated with the endogenous variables they are instrumenting: $\log(FR_{it})$ and $(\log(FR_{it}) \times \log(Y_{it}))$. In a two-stage least squares (2SLS) setup, this is tested in the first-stage regression, where the endogenous variables are regressed on the instruments and all exogenous controls. The first-stage results show highly statistically significant coefficients for $\log(FR_{it-2})$ and $(\log(FR_{it-2}) \times \log(Y_{it}))$, satisfying the relevance condition. This is plausible, as past fertility rates are typically strongly correlated with current fertility due to persistence in demographic trends, and the interaction with $\log(Y_{it})$ reflects how past fertility may moderate current economic conditions.

Fixed Effects and Year Effects

In addition, when fixed effects are included, they absorb time-invariant unobserved characteristics (e.g., cultural norms or institutional factors) that could otherwise create a correlation between the instruments and the error term. By controlling for these factors, fixed effects strengthen the case that the instruments satisfy the exogeneity condition, as the instruments are less likely to pick up the effects of these confounding variables. Also, the first-differencing of the dependent variable eliminates fixed effects implicitly. However, explicitly including fixed effects in the model provides an additional layer of control for time-invariant heterogeneity. Moreover, first-differencing can introduce serial correlation in the errors, which might complicate instrument validity. Using instruments like the second lag of fertility (rather than the first lag) can help avoid this issue, and fixed effects further reinforce the argument by ensuring that any remaining persistent factors are accounted for.

As for the year effects:

- Year effects capture factors that impact all units (e.g., countries) similarly at a given point in time. These could include global events like economic recessions, pandemics, or widespread policy shifts. By accounting for these common shocks or trends, time dummies reduce the risk that unobserved time-specific factors influence both the dependent variable and the endogenous variables. This isolation helps ensure that the instruments are not picking up these omitted effects, making them more likely to be valid.

- Strengthening Instrument Exogeneity: For instruments to be valid, they must be uncorrelated with the error term in the model. Without time dummies, a time-specific shock (e.g., a global downturn) could simultaneously affect the endogenous variable (say, fertility rates) and the outcome variable (say, infant mortality), creating a correlation between the instruments and the error term. Time dummies absorb these shocks,

minimizing such correlations and strengthening the argument that the instruments are exogenous—i.e., not influenced by the same factors driving the error term.

- Working Alongside Fixed Effects: While fixed effects in a panel data model control for unobserved differences across units that do not change over time (e.g., cultural norms), time dummies address unobserved factors that vary over time but are common across units. Together, they provide a robust defense against omitted variable bias. This dual approach ensures that the variation captured by the instruments is less likely to reflect unaccounted-for influences, further supporting their validity.

- Reducing Serial Correlation and Time Trends: Panel data often exhibit serial correlation or trends in the error term, which can complicate instrument validity by violating assumptions of independence over time. Time dummies help by capturing common patterns or cycles (e.g., economic booms and busts), reducing residual serial correlation. This is especially important when instruments rely on lagged variables, as it increases confidence that these lagged instruments are not correlated with the current error term.

Exogeneity of the Instrument

The instruments must be uncorrelated with the error term u_{it} in the structural equation. This means that $\log(FR_{it-2})$ and $(\log(FR_{it-2}) \times \log(Y_{it}))$ should not have a direct effect on $\Delta \log(IMR_{it})$ except through their influence on the endogenous variables $\log(FR_{it})$ and $(\log(FR_{it}) \times \log(Y_{it}))$. This exogeneity condition ensures that the instruments affect infant mortality only through fertility and its interaction with economic development, rather than through any omitted variables or external shocks. The validity of this assumption is evaluated through the following arguments:

- **Timing and Predetermination**

  - Second Lag of Log Fertility Rate ($\log(FR_{it-2})$): This variable is determined at time $t-2$, two periods before the current period t. In a panel data context, it is predetermined, meaning it depends only on information and decisions made prior to t. If u_{it} represents contemporaneous shocks to the change in infant mortality at t (e.g., unexpected health crises), these shocks cannot influence fertility at $t-2$. For $\log(FR_{it-2})$ to be invalid, it would need to directly affect $\Delta \log(IMR_{it})$ independently of $\log(FR_{it})$. However, once $\log(FR_{it})$ and other controls are included, any effect of past fertility on infant mortality changes should be mediated through current fertility, leaving $\log(FR_{it-2})$ uncorrelated with u_{it}.

– Interaction Term ($\log(FR_{it-2}) \times \log(Y_{it})$): This instrument combines the predetermined $\log(FR_{it-2})$ with the current $\log(Y_{it})$, which is a control variable assumed to be exogenous (i.e., $\text{Cov}(\log(Y_{it}), u_{it}) = 0$). The question is whether this interaction is correlated with u_{it}. Since $\log(FR_{it-2})$ is fixed at $t-2$ and unaffected by u_{it}, and $\log(Y_{it})$ is exogenous, their product should not be influenced by contemporaneous shocks in u_{it}, provided there are no omitted variables or unmodeled dynamic effects linking past fertility and current GDP per capita to infant mortality changes directly.

- **Assumption of No Serial Correlation in Errors**

  In panel data regressions, particularly those using first-differenced variables such as $\Delta \log(IMR_{it})$ to eliminate unobserved fixed effects, it is often assumed that the error term ϵ_{it} is not serially correlated, i.e., $\text{Cov}(\epsilon_{it}, \epsilon_{i,t-2}) = 0$. If this assumption holds, $\log(FR_{it-2})$, which is determined at $t-2$, should be uncorrelated with ϵ_{it}, as past fertility cannot be influenced by current or future shocks. Similarly, since $\log(Y_{it})$ is controlled for and assumed to be exogenous, the interaction term ($\log(FR_{it-2}) \times \log(Y_{it})$) should also remain uncorrelated with ϵ_{it}. This reinforces the validity of both instruments, ensuring that they are not driven by contemporaneous disturbances in the error term.

3.2 First-stage Regression

Since fertility may be endogenous, an instrumental variable (IV) approach is employed, using lagged fertility rates as instruments. The first-stage regressions are specified as follows:

$$\begin{aligned}\log(FR)_{it} = \pi_1 \log(FR)_{it-2} + \pi_2(\log FR_{it-2} \times \log Y_{it}) + \pi_3 \log(Y_{it}) \\ + \pi_4 \Delta \log(Y_{it}) + \pi_5 \log(U_{it}) + \sum_t \pi_{6t} \text{Year}_t + u_i + v_{it}\end{aligned} \tag{2}$$

$$\begin{aligned}(\log FR \times \log Y)_{it} = \theta_1 \log(FR)_{it-2} + \theta_2(\log FR_{it-2} \times \log Y_{it}) + \theta_3 \log(Y_{it}) + \theta_4 \Delta \log(Y_{it}) \\ + \theta_4 \log(U)_{it} + \sum_t \theta_{5t} \text{Year}_t + z_i + w_{it}\end{aligned} \tag{3}$$

In these equations, $\log(FR)_{it-2}$ represents the second lag of the fertility rate, which serves as an instrument. This choice is motivated by the assumption that past fertility decisions

influence current fertility rates but are unlikely to be directly affected by contemporaneous shocks to infant mortality. Additionally, the interaction term ($\log FR_{it-2} \times \log Y_{it}$) captures how economic conditions moderate the persistence of fertility, ensuring that the instrument varies with macroeconomic factors.

The inclusion of fixed effects, u_i and z_i, is crucial since fertility appears in levels rather than differences. These fixed effects account for unobserved country-specific heterogeneity that may influence fertility and mortality simultaneously, controlling for persistent cultural, institutional, or structural differences across countries. The terms v_{it} and w_{it} represent the first-stage error components, capturing idiosyncratic shocks. Furthermore, $\sum_t \text{Year}_t$ accounts for time trends and period-specific factors that may systematically affect fertility and mortality over time.

3.3 Second-Stage Regression

With the first-stage regressions confirming that the instrument is strongly correlated with fertility, the second-stage regression leverages these predicted values to estimate the causal effect of fertility on infant mortality. By using the predicted values of fertility ($\log(\hat{FR}_{it})$) and its interaction term ($(\log(\widehat{FR_{it}) \times \log(Y_{it})})$) from the first-stage regressions, this approach ensures that the estimated coefficients reflect exogenous variation in fertility, mitigating potential biases arising from reverse causality and omitted variables. The second-stage equation is specified as follows:

$$\begin{aligned}\Delta \log(IMR_{it}) = \beta_0 + \beta_1 \log(\hat{FR}_{it}) + \beta_2(\log(\widehat{FR_{it}) \times \log(Y_{it})}) + \beta_3 \log(Y_{it}) \\ + \beta_4 \Delta \log(Y_{it}) + \beta_5 \log(UrbanPop_{it}) + \sum_t \gamma_t \text{Year}_t + \epsilon_{it} \qquad (4)\end{aligned}$$

The second-stage regression provides an unbiased estimate of the effect of fertility on infant mortality by isolating exogenous variation in fertility. By accounting for fixed effects, year-specific shocks, and key economic controls, this approach ensures that the relationship between fertility and infant mortality is not confounded by unobserved heterogeneity or time-varying factors. The inclusion of the interaction term between fertility and GDP per capita allows for a more nuanced analysis, capturing how economic development influences the magnitude of the fertility-mortality relationship. The estimated coefficients from this stage offer valuable insights into the dynamics between demographic and economic factors, reinforcing the importance of economic conditions in shaping the impact of fertility on child survival rates.

4 Data

This study examines the socio-economic determinants of infant mortality using a panel dataset covering over 200 countries from 1960 to 2023. The dataset is primarily sourced from the *World Bank's World Development Indicators*, except for GDP per capita, which is obtained from the *Penn World Table* (measured in constant 2017 USD). The dataset includes key macroeconomic and demographic indicators relevant to infant mortality.

The panel dataset consists of 8,492 country-year observations, but it is unbalanced due to variations in data availability across countries and time. Some countries have complete or near-complete time series, while others have intermittent missing data. The number of observations per country ranges from a minimum of 5 to a maximum of 63, with an average of approximately 42.3 observations per country. This unbalanced nature arises from historical data inconsistencies, differences in reporting practices, and country-specific events that affect data collection. The empirical strategy accounts for this imbalance to ensure robustness in estimation.

The key variables used in the analysis are defined as follows:

- **Infant Mortality Rate (IMR_{it})**: The number of deaths among children under one year of age per 1,000 live births. This serves as the primary dependent variable.
- **Fertility Rate (FR_{it})**: The total fertility rate, representing the average number of children a woman is expected to have over her lifetime.
- **Real GDP per Capita (Y_{it})**: The inflation-adjusted economic output per person in a country, reflecting living standards and overall economic prosperity.
- **Urban Population Share (U_{it})**: The percentage of the population residing in urban areas, serving as a proxy for healthcare access, infrastructure, and sanitation.
- **Growth in IMR ($\Delta \log(IMR_{it})$)**: The log difference in infant mortality, capturing the percentage change in child mortality rates over time.
- **Economic Growth ($\Delta \log(Y_{it})$)**: The log difference in real GDP per capita, representing short-term changes in economic development.

Table 1 (see Appendix) presents summary statistics for the key variables used in the analysis. The mean infant mortality rate is approximately 47.81 per 1,000 live births, with significant variation across countries (standard deviation of 43.01), highlighting persistent disparities in child survival rates. Fertility rates also show considerable variation, ranging

from as low as 0.70 to as high as 8.86 children per woman, suggesting differences in demographic trends and access to reproductive healthcare.

Economic conditions vary widely, with real GDP per capita ranging from 236.73 to 263,463.4 USD, reflecting stark contrasts between low-income and high-income nations. Similarly, urbanization rates range from just 2% to 100%, reinforcing the heterogeneity in urban infrastructure and healthcare access across countries. The annual growth in IMR and economic growth indicators exhibit relatively small mean values but display significant variation, indicating substantial shifts in economic and health outcomes across time and regions.

These statistics underscore the importance of considering both level and growth dynamics in explaining variations in child mortality.

5 Results and Analysis

This section presents the empirical findings from the instrumental variable regression model, which examines the relationship between fertility and infant mortality while accounting for potential endogeneity. Using a two-stage least squares (G2SLS) approach, the analysis provides robust estimates by leveraging lagged fertility rates and their interaction with economic development as instrumental variables. The results underscore the significant role of demographic and economic factors in shaping infant mortality outcomes over time.

5.1 First-Stage Regression Results

The first-stage regression confirms the strength of the chosen instruments, demonstrating that lagged fertility $\log(FR_{it-2})$ and its interaction with GDP per capita $\log(FR_{it-2}) \times \log(Y_{it})$ are significantly correlated with current fertility and its interaction with economic conditions. The coefficient on $\log(FR_{it-2})$ is positive and highly significant ($\pi_1 = 1.161$, $p < 0.001$), indicating strong persistence in fertility behavior over time. This result aligns with demographic theories suggesting that fertility decisions are influenced by long-term socioeconomic patterns and cultural norms.

The interaction term $\log(FR_{it-2}) \times \log(Y_{it})$ is negative and significant ($\pi_2 = -0.023$, $p < 0.001$), implying that past fertility, when coupled with economic development, contributes to lower current fertility. This reflects the moderating effect of income: in wealthier settings, fertility declines over time due to improved access to education, family planning services, and employment opportunities, particularly for women.

Among the control variables, the coefficient on urban population share $\log(U_{it})$ is negative and statistically significant ($\pi_5 = -0.0228$, $p < 0.01$), consistent with expectations that

urban areas offer better access to healthcare, education, and contraception, leading to lower fertility. Meanwhile, the coefficient on real GDP per capita $\log(Y_{it})$ is positive and significant ($\pi_3 = 0.0395$, $p < 0.001$), which may appear counterintuitive at first glance. However, this does not imply that wealthier countries have higher fertility rates. Rather, this positive coefficient captures short-run variation in the context of a model that also includes the interaction term. When interpreted alongside the negative interaction effect, it becomes clear that the influence of income on fertility is conditional: at lower levels of GDP per capita, increases in income may initially coincide with rising fertility due to improved child survival, but this effect diminishes or reverses at higher income levels. This nuanced relationship is consistent with prior literature on demographic transitions.

Economic growth $\Delta \log(Y_{it})$ is positively associated with fertility ($\pi_4 = 0.0199$, $p = 0.071$), though marginally significant. This may suggest that in the short run, income gains can relax budget constraints and temporarily raise fertility intentions, particularly in developing contexts where family size remains economically motivated.

5.2 Second-Stage Regression Results

The second-stage regression results provide strong empirical evidence linking fertility rates to infant mortality. The estimated coefficient on $\log(FR_{it})$ is positive and highly significant ($\theta_1 = 0.0997$, $p < 0.001$); however, its interpretation is conditional on the interaction term ($\log(FR_{it}) \times \log(Y_{it})$). The negative and statistically significant coefficient on this interaction term ($\theta_2 = -0.0106$, $p < 0.001$) suggests that the effect of fertility on infant mortality diminishes as income levels rise. In lower-income settings, higher fertility is more strongly associated with increased infant mortality, likely due to resource constraints, reduced maternal care, and limited healthcare access. However, as economic development progresses, these adverse effects are mitigated, as wealthier economies benefit from better healthcare infrastructure, improved maternal education, and increased investments in child health.

The coefficient on $\log(Y_{it})$ is positive and statistically significant ($\theta_3 = 0.0154$, $p < 0.001$), which might initially seem counterintuitive since higher GDP per capita is generally expected to reduce infant mortality. However, when considered in conjunction with the interaction term, the interpretation changes. At lower fertility levels, economic development can coincide with increased mortality due to transitional health risks, urbanization pressures, or epidemiological shifts, such as the rise in non-communicable diseases and environmental pollution. In high-fertility settings, the interaction term implies that the negative impact of GDP per capita on mortality becomes more pronounced, emphasizing the role of economic growth in improving child health outcomes.

To better understand the relationship between fertility, national income, and infant mortality, the marginal effect of fertility is calculated for a country with the median income level. The marginal effect of fertility is approximately 0.0241, indicating that for a country with median income, a one-unit increase in the fertility rate corresponds to a 2.4% increase in the log of infant mortality. This result underscores that while fertility positively impacts infant mortality, the effect is conditional on the income level.

Additionally, the marginal effect of national income is calculated for a country with the median fertility rate. The marginal effect is approximately 0.0031, meaning that a one-unit increase in national income corresponds to a 0.31% increase in the log of infant mortality. This indicates that while national income is associated with higher infant mortality, this effect is conditional on fertility levels, with higher fertility amplifying the impact of income on mortality outcomes.

The control variables further reinforce these findings. Short-term economic growth, $\Delta \log(Y_{it})$, is negatively associated with infant mortality ($\theta_4 = -0.0477$, $p < 0.001$), suggesting that economic expansions contribute to better child survival rates. This effect is likely mediated through increased public spending on healthcare, improvements in sanitation and infrastructure, and rising household incomes. Additionally, urbanization is negatively associated with infant mortality ($\theta_5 = -0.0089$, $p < 0.01$), highlighting the role of improved healthcare accessibility, better sanitation, and increased maternal education in reducing child mortality rates.

By considering both the direct effects and interaction effects, these results provide a nuanced understanding of how fertility and economic development jointly shape infant mortality outcomes. The findings emphasize the importance of policy interventions that simultaneously address fertility rates and economic conditions to maximize child health improvements.

5.3 Statistical Inference and Robustness

To ensure statistical inference accounts for within-country correlation over time, standard errors are clustered at the country level. This adjustment addresses three key econometric concerns. First, it accounts for *heteroskedasticity*, where variance differs across countries due to variations in healthcare quality, economic structure, and demographic factors. Second, it corrects for *serial correlation*, ensuring that country-specific trends over time do not bias the results. Finally, clustering prevents *overstating statistical significance* by ensuring that observations within the same country are not treated as independent. This is particularly important in a panel setting where repeated measurements of the same units may lead to correlated error terms.

Additionally, the robustness of the model is supported by the within-group R^2 value of 0.0787, which, while relatively modest, is consistent with expectations given that the model relies on first-differenced dependent variables. First differencing reduces the total variation available for explanation, which often results in lower R^2 values. However, despite the modest fit, the explanatory variables still account for a meaningful proportion of the variation in infant mortality. Moreover, the Wald test statistic ($\chi^2(62) = 413.21, p < 0.001$) confirms the joint significance of the model's regressors, reinforcing the robustness of the estimates.

6 Discussion

The empirical findings provide significant insights into the relationship between fertility, economic development, and infant mortality. The results from the instrumental variable estimation confirm that higher fertility rates are positively associated with infant mortality, while economic development plays a moderating role in this relationship. In this section, we discuss the economic mechanisms underlying these results and interpret them in the context of demographic transitions, healthcare accessibility, and resource constraints.

6.1 The Role of Fertility in Infant Mortality

The second-stage regression results indicate that an increase in fertility rates is significantly associated with higher infant mortality, as shown by the positive coefficient on $\log(FR_{it})$. This relationship aligns with economic theories of household resource allocation. In settings with high fertility, parents must allocate limited time and financial resources across a greater number of children, which can reduce per-child investment in health, nutrition, and maternal care. As a result, the probability of infant mortality increases due to inadequate healthcare access, lower birth weight, and higher risks of infectious diseases.

However, economic development appears to mitigate the adverse effects of fertility on infant mortality. The negative and significant coefficient on the interaction term ($\log(FR_{it}) \times \log(Y_{it})$) suggests that as real GDP per capita increases, the detrimental impact of fertility on infant mortality declines. This can be attributed to several factors: higher income levels allow households to invest more in child healthcare, improved maternal nutrition, and access to better medical facilities. Additionally, economic growth enables governments to expand public health infrastructure, increasing vaccination rates and maternal health programs.

These findings are visually confirmed in Figure 1 in the appendix, which plots the marginal effect of fertility on infant mortality across different fertility levels. The decreasing trend in marginal effects suggests that while higher fertility is initially associated with

increased infant mortality, its effect diminishes as economic conditions improve.

6.2 Economic Development and Infant Health

The results also highlight the role of economic growth in improving child survival rates. The coefficient on $\log(Y_{it})$ is positive, suggesting that higher levels of economic development are initially associated with higher infant mortality. This counterintuitive result can be explained by demographic and epidemiological transitions: early stages of economic growth are often accompanied by increased urbanization, pollution, and transitional health risks, leading to higher mortality rates despite rising income levels. However, the positive coefficient on $\log(Y_{it})$ should be interpreted in conjunction with the interaction term between fertility and income. The presence of the interaction term indicates that the effect of national income on infant mortality is conditional on fertility rates. Specifically, in high-fertility contexts, the positive effect of economic development on mortality is mitigated.

When examining the effect of income growth ($\Delta \log(Y_{it})$), we find a significant negative impact on infant mortality. This indicates that short-term economic expansions lead to better child health outcomes, likely through increased public spending on healthcare, improved household income, and infrastructure development.

The non-linear relationship between economic development and infant mortality is further illustrated in Figure 2. The marginal effect of $\log(Y_{it})$ on infant mortality is positive at lower levels of income but turns negative as GDP per capita rises. This finding aligns with the Preston Curve, which suggests that beyond a certain income threshold, economic growth leads to substantial improvements in child survival by enhancing healthcare infrastructure, sanitation, and education.

6.3 Urbanization and Demographic Transition

Urbanization is another key factor influencing infant mortality. The negative coefficient on $\log(U_{it})$ suggests that higher urban population shares are associated with lower infant mortality. This finding is consistent with economic theories of agglomeration, where urban areas benefit from economies of scale in healthcare provision, better medical facilities, and improved maternal education. However, rapid urbanization without corresponding improvements in infrastructure can lead to overcrowding and increased disease transmission, potentially offsetting some of these benefits.

6.4 Policy Implications

The findings have important policy implications. First, efforts to reduce infant mortality should focus on economic development strategies that promote sustainable income growth, as short-term income fluctuations appear to have a strong influence on child health. Second, family planning and maternal health programs should be prioritized in high-fertility regions, particularly where economic growth remains low. The results suggest that interventions aimed at reducing fertility,such as increased female education, access to contraception, and healthcare improvements, could play a vital role in lowering infant mortality rates. Finally, targeted policies in urban areas, such as investments in sanitation and healthcare accessibility, can further contribute to improved child health outcomes.

7 Conclusion

This study examines the relationship between fertility, economic development, and infant mortality, providing empirical evidence on how macroeconomic conditions shape child survival outcomes. Using a two-stage least squares (2SLS) approach, the analysis addresses endogeneity concerns by instrumenting fertility rates and their interaction with economic conditions. The findings indicate that higher fertility rates are associated with increased infant mortality, but this effect diminishes as real GDP per capita rises. Economic growth enables greater investments in healthcare, improved maternal nutrition, and expanded public health infrastructure, mitigating the adverse effects of high fertility on child mortality. This non-linear relationship aligns with the Preston Curve hypothesis, which suggests that beyond a certain income threshold, economic progress leads to substantial improvements in child survival.

The results also highlight the significance of income growth in improving infant health outcomes. While initial stages of economic development may coincide with transitional health risks, short-term income growth has a significant negative effect on infant mortality. This suggests that rising household incomes and increased public spending on healthcare contribute to improved child survival rates over time. Urbanization further emerges as a crucial determinant, as a higher share of urban population is associated with lower infant mortality, likely due to better access to healthcare, sanitation, and education. However, rapid and unplanned urbanization could offset these benefits by exacerbating overcrowding and disease transmission, emphasizing the need for targeted policy interventions.

Despite its contributions, this study has certain limitations. Data constraints may limit the generalizability of the findings, particularly in low-income countries where informal

economies and weak institutions could distort economic indicators. While instrumenting fertility with lagged values addresses endogeneity concerns, potential dynamic feedback effects and omitted variable bias cannot be entirely ruled out. Additionally, the analysis focuses primarily on economic and demographic factors, whereas institutional quality, healthcare policies, and cultural determinants may also play a role in shaping infant mortality trends.

The findings suggest that efforts to reduce infant mortality should emphasize policies that promote sustainable economic growth, improved maternal and child healthcare, and targeted family planning programs in high-fertility regions. Investments in urban infrastructure and healthcare accessibility will also be crucial in ensuring that the benefits of economic development reach all segments of the population. By addressing these factors, countries can accelerate reductions in infant mortality and improve overall public health outcomes.

8 Use of Generative AI and AI-assisted tools

During the preparation of my thesis, I used ChatGPT for assistance with some LaTeX formatting, checking consistency in equations and tables, improving clarity in complex sentences. After using this tool/service, I reviewed and edited the content as needed and take full responsibility for the content of my thesis.

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Appendix

Figure 1: Infant mortality rate trends by income level. Data Source: World Bank.

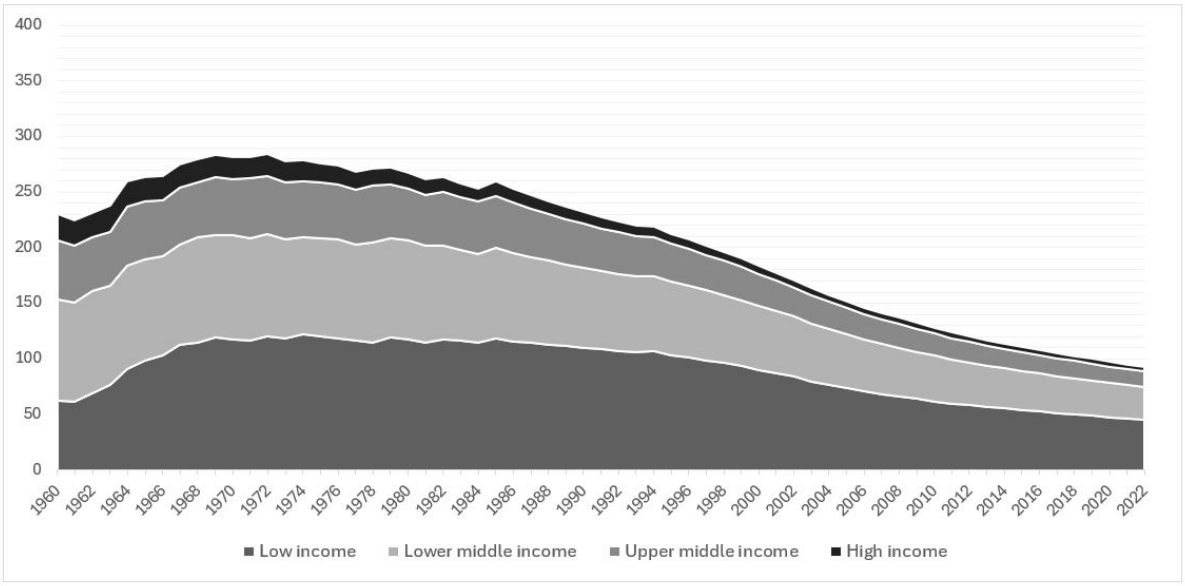

Figure 2: Marginal effect of log Real GDP per capita on infant mortality, with 95% confidence intervals

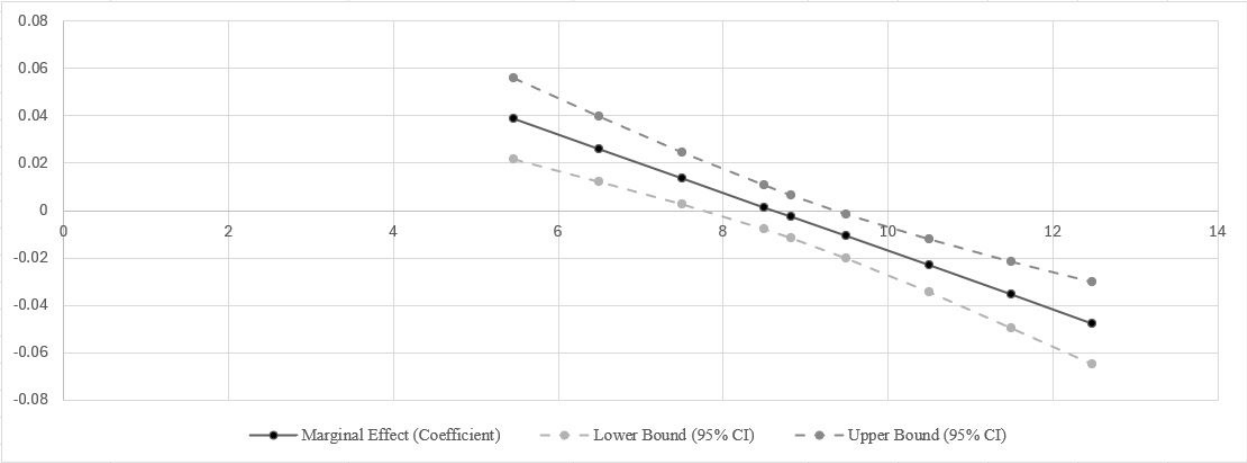

Figure 3: Marginal effect of log fertility rate on infant mortality, with 95% confidence intervals.

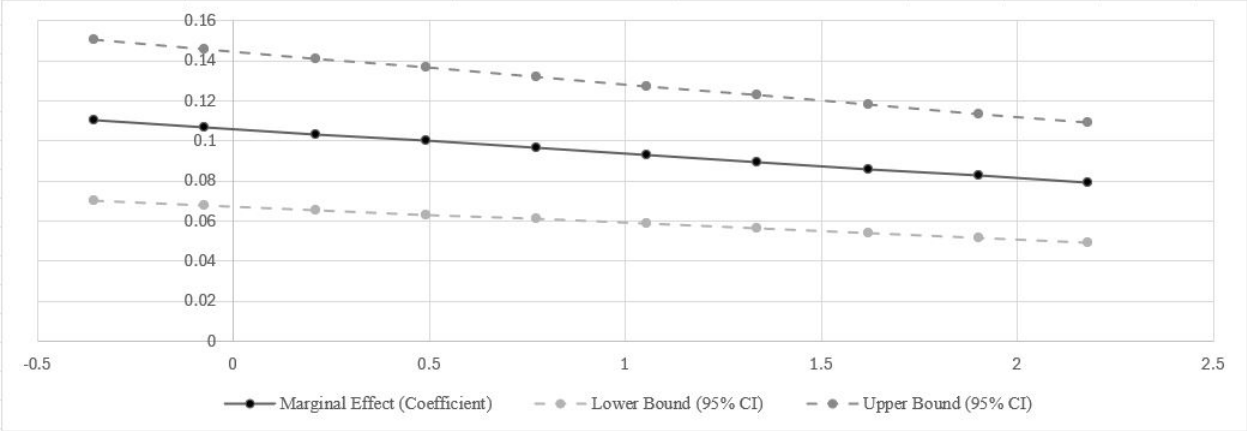

Table 1: Summary Statistics of Key Variables

Variable	**Observations**	**Mean**	**Std. Dev.**	**Min**	**Max**
Infant Mortality Rate (IMR_{it})	8492	47.81	43.01	1.3	272
Fertility Rate (FR_{it})	8492	3.89	1.96	0.70	8.86
Real GDP per Capita (Y_{it})	8492	13,844.01	19,797.06	236.73	263,463.4
Urban Population Share (U_{it})	8492	50.37	24.77	2.08	100
Growth in IMR ($\Delta \log(IMR_{it})$)	8492	-0.032	0.0313	-0.9033	0.7162
Economic growth ($\Delta \log(Y_{it})$)	8492	0.0216	0.0855	-1.1678	0.8869

Note: Summary statistics include the number of observations, mean, standard deviation, minimum, and maximum values for each key variable.

Table 2: First-Stage Regression Results for Fertility Rate ($\log FR_{it}$)

Variable	**Coefficient**	**Std. Error**	**p-value**
Dependent Variable: Fertility Rate ($\log FR_{it}$)			
Real GDP per Capita ($\log Y_{it}$)	0.0395	0.0056	0.000
Economic Growth ($\Delta \log Y_{it}$)	0.0199	0.0110	0.071
Urban Population Share ($\log U_{it}$)	-0.0228	0.0082	0.006
Instrument: Lagged Fertility Rate ($\log FR_{it-2}$)	1.1613	0.0389	0.000
Instrument: Interaction Term ($\log FR_{it-2} \times \log Y_{it}$)	-0.0230	0.0045	0.000
Observations	8,492		
Wald χ^2	5,490		0.000

Note: This table presents the first-stage regression results where the dependent variable is the fertility rate ($\log FR_{it}$). Standard errors are reported in parentheses.

Table 3: First-Stage Regression Results for Interaction Term ($\log FR_{it} \times \log Y_{it}$)

Variable	**Coefficient**	**Std. Error**	**p-value**
Dependent Variable: Interaction Term ($\log FR_{it} \times \log Y_{it}$)			
Real GDP per Capita ($\log Y_{it}$)	0.3350	0.0508	0.000
Economic Growth ($\Delta \log Y_{it}$)	0.1861	0.1000	0.063
Urban Population Share ($\log U_{it}$)	-0.1804	0.0702	0.010
Instrument: Lagged Fertility Rate ($\log FR_{it-2}$)	1.5715	0.3523	0.000
Instrument: Lagged Interaction Term ($\log FR_{it-2} \times \log Y_{it}$)	0.7840	0.0419	0.000
Observations	8,492		
Wald χ^2	621		0.000

Note: This table presents the first-stage regression results for the interaction term. Standard errors are reported in parentheses.

Table 4: Second-Stage 2SLS Regression Results

Variable	**Coefficient**	**Std. Error**	**p-value**
Dependent Variable: $\Delta \log IMR_{it}$			
Fertility Rate ($\log FR_{it}$)	0.0997	0.0153	0.000
Interaction Term ($\log FR_{it} \times \log Y_{it}$)	-0.0107	0.0017	0.000
Real GDP per Capita ($\log Y_{it}$)	0.0154	0.0021	0.000
Economic Growth ($\Delta \log Y_{it}$)	-0.0477	0.0124	0.000
Urban Population Share ($\log U_{it}$)	-0.0089	0.0028	0.001
Observations	8,492		
R^2 (Overall)	0.0889		
Wald χ^2	413.21		0.000

Note: This table reports the second-stage results from the 2SLS regression, where the fertility rate ($\log FR_{it}$) and its interaction with real GDP per capita ($\log Y_{it}$) are instrumented using their respective lagged values. Standard errors are reported in parentheses.