Who Gets Cared For and Why? Fertility Shocks, Intra-Household Investment Reallocation, and the Quantity–Quality Trade-Off in the Philippines

By Erika Salvador
Caroline Theoharides*

This paper investigates how increases in fertility, driven by reduced access to contraception, affect parental investment in child health. I use Executive Order No. 003—a 2000 policy in Manila that restricted access to modern contraceptives—as an exogenous fertility shock. Drawing on pooled data from the 1998, 2003, and 2008 Philippine Demographic and Health Surveys, I estimate two-stage least squares models that use policy exposure to instrument for sibship size. Child health inputs fall into two categories: prenatal (proxied by birth weight) and postnatal (measured by the total number of vaccine doses received by age five). The first-stage results indicate that affected mothers had, on average, 0.20 more living children at the time of a birth. In the second stage, each additional sibling is associated with a reduction of 22.97 vaccine doses received by the index child, a decline large enough to prevent completion of the recommended immunization schedule. The estimated effect on birth weight is −8,676 grams, though statistically insignificant Reduced-form estimates are directionally consistent: policy exposure is associated with a 0.91-dose drop in vaccine uptake and a 349.60-gram decrease in birth weight. While instrumental variable estimates are limited by a weak first stage, the magnitude and sign of the coefficients support the hypothesis that larger sibship sizes reduce health investments per child. The results suggest that postnatal investments, particularly those requiring sustained parental effort, are more sensitive to fertility shocks than prenatal outcomes.

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I. Introduction

The trade-off between child quantity and child quality is a foundational concept in the economics of the family. First articulated by Becker (1960) and extended in subsequent models of household behavior (Becker and Lewis, 1973; Becker and Tomes, 1976), this framework posits that parents allocate finite resources—both financial and non-financial—across children. An increase in fertility dilutes the resources available per child and, under binding constraints, may lead to reduced investments in each child's human capital.

Empirical investigations of the quantity-quality trade-off have focused primarily on educational outcomes. Most studies examine how increases in maternal fertility—or equivalently, the number of siblings a child has—affect school enrollment, grade progression, test scores, and completed years of schooling (Rosenzweig and Wolpin, 1980; Black, Devereux and Salvanes, 2005; Angrist, Lavy and Schlosser, 2010). These outcomes serve as accessible proxies for long-run human capital accumulation, but they represent only one dimension of child quality. Other dimensions of child quality–such as health at birth and early childhood health investments—are early-onset or biologically constrained, and they tend to be less amenable to remediation later in life. For instance, babies born with very low birth weight face higher risks of infant mortality and long-term developmental deficits that cannot be fully reversed (Cook and Fletcher, 2015). Similarly, a child who misses critical early vaccinations is more vulnerable to infectious diseases that can kill or disable them, which results to lifelong negative health impacts (Costa et al., 2024). Such early-life health setbacks may permanently impair cognitive development and physical growth. This limits long-run productivity regardless of subsequent improvements in schooling or income.

The omission of these health outcomes from much of the empirical literature leaves important aspects of the quantity-quality trade-off unexamined. Investments in maternal and child health—such as adequate prenatal care leading to healthy birth weight and timely childhood immunizations—are important to early development and long-term outcomes (Victora et al., 2008; Hoddinott et al., 2013*a*). They shape brain development, immune system functioning, and physical stature, and they have been shown to predict later-life earnings and health outcomes across a wide range of settings (Grantham-McGregor et al., 2007; Alderman, Hoddinott and Kinsey, 2006). Unlike educational gaps, which may be partially addressed later in life, early health deficits—such as poor nutrition or missed vaccinations—are often irreversible (Victora et al., 2008; Hoddinott et al., 2013*a*). As a result, any credible evaluation of the quantity-quality trade-off must include early-life health outcomes to fully capture the long-term consequences of different fertility shocks.

This study addresses that gap by examining how a localized, exogenous in-

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crease in fertility influenced early-life health outcomes in the Philippines. In 2000, then–Mayor José "Lito" Atienza of Manila City issued Executive Order No. 003, which effectively barred the provision and promotion of modern contraceptives in all city-run health facilities. The directive eliminated access to pills, condoms, intrauterine devices, and other forms of modern family planning, and mandated that public healthcare providers offer only natural methods. The policy remained in effect for nearly a decade and applied solely within the jurisdiction of the Manila city government. No comparable restriction was implemented at the national level, and neighboring cities within Metro Manila continued to provide access to modern contraceptives. This policy created a clear spatial and temporal discontinuity in access to family planning services—one that was plausibly unrelated to changes in fertility preferences, household income, or broader governance trends. As such, the Manila contraceptive ban provides a quasi-experimental shock to fertility exposure among poor urban households.

I use this natural experiment to estimate the causal impact of increased family size on early-childhood health outcomes. The analysis uses nationally representative data from multiple rounds of the Philippine Demographic and Health Survey (DHS), which include detailed information on household structure, maternal characteristics, and child health indicators. The primary outcomes are birth weight (measured in grams, when reported) and whether the child received key early-life vaccinations, including those against polio, diphtheria–pertussis–tetanus (DPT), and measles, as a measure of essential postnatal health investment. These outcomes are widely recognized indicators of child well-being and reflect both biological and behavioral dimensions of parental investment in early development. The empirical strategy follows a difference-in-differences design that compares child outcomes in Manila and comparable urban areas before and after the onset of the policy.

The identification strategy rests on two key assumptions. First, in the absence of the contraceptive ban, trends in child health outcomes in Manila would have evolved in parallel with those in the comparison cities. Second, no other policy changes or economic shocks in Manila during the study period coincided with the timing and scope of the contraceptive policy. I probe these assumptions using falsification tests, placebo comparisons, and robustness checks that include city-specific time trends, mother fixed effects, and controls for baseline demographic differences. The analysis proceeds in three stages. I first replicate existing work (Dumas and Lefranc, 2019) to confirm that the contraceptive ban led to an increase in fertility among affected women. Next, I estimate the reduced-form effects of exposure to the policy on the selected child health outcomes (birth weight, and vaccination uptake). Finally, I examine heterogeneity in these effects across subgroups defined by maternal education, household wealth, and access to prenatal care. These characteristics proxy for a household's resources and its capacity to buffer the impact of increased family size on child health.

This study contributes to the literature in several important ways. It provides

new evidence on how an externally induced increase in family size, resulting from a local restriction on contraceptive access, can affect child health in poor urban communities. Whereas most past research on the quantity-quality trade-off has focused on education, this study broadens the notion of child "quality" to include early-life health measures such as birth weight and immunization status. It also adds to the small number of studies that leverage unexpected changes in reproductive health policy to examine long-term effects on children's well-being. More broadly, the findings indicate that local restrictions on family planning can unintentionally harm children's health, especially in settings where families already face poverty, food insecurity, and limited public health services.

II. Review of Related Literature

The quantity-quality (Q–Q) theory, a central idea in modern family economics, holds that parents face a trade-off between the number of children and the "quality" of investment—such as education or health—they can provide to each. Quality in this context refers to the human capital of each child: attributes like education, health, and nutrition that enhance a child's future productivity and well-being. The genesis of this idea traces back to Gary Becker's seminal work around 1960, which for the first time treated children as economic goods subject to parental choice and budget constraints (Becker, 1960). Becker argued that as families become wealthier, they may not simply want more children, but rather better-raised children, much as a household might prefer a higher-quality car or house over a greater quantity of them. This proposition led to a formal theory in which increases in income or changes in economic conditions cause parents to substitute child quality for quantity, consistent with historical patterns of lower fertility and higher educational attainment during economic development (Galor and Weil, 2000).

In what follows, I review the theoretical foundations of the Q-Q model and its evolution in the literature. I begin with the static models of Becker (Becker, 1960) and Becker–Lewis (Becker and Lewis, 1973), which first formalized the trade-off within a household utility maximization framework. I then examine extensions to dynamic, intergenerational settings, including the contributions of Becker and Tomes (Becker and Tomes, 1976) on child endowments and the altruistic dynastic model associated with Barro and Becker (Barro and Becker, 1989). Next, I turn to macroeconomic and unified growth models, notably Galor and Weil (Galor and Weil, 2000) and Galor and Moav (Galor and Moav, 2002), which integrate the Q–Q mechanism into a general theory of demographic and economic transformation.

Finally, I discuss more recent refinements that enrich the basic model by incorporating credit constraints (Doepke, 2004), intra-household bargaining (Doepke and Kindermann, 2019), and multi-dimensional child quality (Hoddinott et al., 2013*b*; Kalemli-Ozcan, 2002), with a special emphasis on health and nutrition. The literature review focuses on how the Q–Q framework has been applied to understand fertility and child investment patterns, especially in developing country

contexts where resource constraints and health outcomes are paramount.

A. *Theoretical Background*

Becker's Static Model

Becker's early work introduced an economic model of fertility and treated children as durable goods that provide utility to parents but impose costs (Doepke, 2015). In Becker's 1960 model, a household derives satisfaction from the number of children (n) and from the quality of each child (q), alongside conventional consumption of other goods (y). A simple representation is a utility function:

$$U = U(n, q, y),$$

with U increasing in each argument up to some satiation point. Here, quality q can be thought of as the expenditure or investment per child (e.g. education spending, health care, nutrition), assumed for now to be the same for each child. Parents face a budget constraint that links quantity and quality: raising more children dilutes the resources available per child. A prototypical budget constraint (in static form) can be written as:

$$p_y y + p_n n + p_q nq = I,$$

where I is total family income (or full income), p_n represents baseline, non-discretionary costs associated with each additional child (e.g. expenditures on food, shelter, or clothing that are incurred irrespective of quality-enhancing investments), and p_q denotes the marginal cost of investing in one unit of quality per child.The term $p_q nq$ captures total expenditure on quality for all children and is linear in n. As the number of children rises, parents must extend any chosen level of q across a broader base, which amplifies the total cost of quality. Conversely, the term $p_n n$ implies that the cost of an additional child rises with the quality level q already chosen, since each child must meet a higher standard of care or investment. For instance, a household that chooses to provide more education or better health care per child incurs an additional burden when it expands family size, as each child must receive the same enhanced level of investment. Similarly, a larger family increases the cumulative cost of quality, even if q remains fixed, due to the need to replicate expenditures across more children. In short, the shadow price of child quality increases with n, and the shadow price of child quantity increases with q. The cost structure induces a mutual dependence between quantity and quality, such that any adjustment along one dimension alters the effective cost of the other.

Becker and Lewis (1973) formalize the mutually reinforcing nature of the quantity-quality cost structure. An increase in n raises the total cost required to sustain a given level of q for each child, while a higher level of q raises the marginal cost associated with having an additional child. For example, allocating more resources to education or health per child increases the financial burden of

expanding family size. This interdependence links the two decisions directly. The household cannot choose n and q in isolation; each choice alters the marginal cost of the other.

Mathematically, the trade-off appears in the first-order conditions of the household's optimization problem. Let λ represent the Lagrange multiplier on the full-income constraint.

$$\mathcal{L} = U(n, q, y) + \lambda \left(I - p_y y - p_n n - p_q n q \right).$$

The first-order conditions are:

$$\frac{\partial \mathcal{L}}{\partial n} = U_n - \lambda(p_n + p_q q) = 0, \quad \frac{\partial \mathcal{L}}{\partial q} = U_q - \lambda p_q n = 0, \quad \frac{\partial \mathcal{L}}{\partial y} = U_y - \lambda p_y = 0.$$

Combining the first two yields:

$$\frac{U_n}{U_q} = \frac{p_n + p_q q}{p_q n}.$$

This condition equates the marginal rate of substitution between quantity and quality to the ratio of their full marginal costs. The numerator rises with q, and the denominator rises with n. As one choice increases, the relative cost of the other becomes higher. This relationship induces substitution toward the less costly dimension. The trade-off between quantity and quality arises from the structure of the budget itself. It does not rely on specific assumptions about utility curvature or intrinsic substitutability (Becker and Lewis, 1973).

This formulation implies two core predictions. Firstly, although both child quantity and child quality may rise with income, the household's budget constraint can generate a negative relationship between income and fertility. As income increases, total spending on children tends to rise, but the allocation often favors quality over quantity. Becker illustrated this with the analogy of durable goods: wealthier households tend to upgrade the quality of a house or a car rather than acquire additional units. In a similar way, higher-income families often direct additional resources toward education, nutrition, or health per child. Within the model, an income increase ($\mathrm{d}I > 0$) produces a direct effect that makes children more affordable and an indirect effect that discourages fertility. As q rises, the shadow price of an additional child also rises. If the marginal utility from higher quality exceeds that from larger family size, then the substitution effect outweighs the income effect, leading to a lower optimal n. This mechanism offers a structural explanation for the demographic transition: fertility tends to fall as households become richer, even when preferences remain unchanged.

Furthermore, a similar logic applies to changes in the cost parameters p_q and p_n. A decline in p_q, such as through a policy that lowers the price of education or health care, increases q and raises the marginal cost of quantity. This effect reduces optimal fertility. A rise in p_n, which may reflect higher child-rearing

costs or a greater opportunity cost of parental time, reduces the appeal of larger families and can shift resources toward child quality. These outcomes follow from the structure of the budget constraint, without requiring any explicit preference for quality over quantity. Becker and Lewis noted that these comparative static results align with observed patterns. For example, increases in women's wages often reduce fertility more than they reduce educational spending per child. This asymmetry reflects the model's central feature: quantity and quality are linked through their cost structure. An increase in one raises the marginal cost of the other. The model explains how households make trade-offs between the number of children and investments in each.

Intergenerational Models: Altruism and Child Endowments

While the early Q–Q models were static (one-period) representations, subsequent contributions extended the framework to consider fertility and child investment over multiple periods or even multiple generations. The main development in this literature was the incorporation of intergenerational human capital dynamics, where parents derive utility not only from the number and quality of children in the present, but also from the long-run outcomes of their offspring. These extensions allowed child quality to evolve endogenously across time, rather than being determined solely within a single period.

One of the earliest and most influential models of this kind was proposed by Becker and Tomes (1976). In their formulation, each child enters the world with an exogenous endowment E, which may reflect factors such as cognitive ability (e.g., measured IQ or language acquisition speed), early health status (e.g., birth weight or incidence of neonatal complications), genetic predispositions (e.g., risk for chronic illness, temperament, or neurodevelopmental traits), or family background characteristics (e.g., parental education, household stability, or neighborhood conditions). Furthermore, parents can augment this endowment by investing resources q in the form of education, nutrition, and other quality-enhancing inputs. The effective adult human capital of the child might be expressed as $H = E + f(q)$ (in a simple additive form) or a multiplicative variant $H = E \cdot f(q)$, where $f(q)$ is an increasing concave function of parental investment.

Becker and Tomes (1976) emphasized that variation in endowments E can shape how parents allocate investments q across children. When the productivity of investment increases with endowment, parents may concentrate resources on children with higher E, who are more likely to convert additional investment into future success. In other cases, parents may attempt to compensate for lower endowments by directing greater investment toward disadvantaged children. Put simply, child-specific variation in initial conditions affects not only outcomes but also strategic parental choices. Because of this, the relationship between income and the demand for child quality is not uniform. The income elasticity of demand for quality may differ across households, depending on the distribution of endowments within the family and across the broader population.

Furthermore, Becker and Tomes (1976) showed that at low income levels, much of what constitutes child quality comes from exogenous endowments, i.e., factors like public education, neighborhood environment, or access to basic healthcare that are not privately purchased. In these settings, small increases in parental income may not lead to significant changes in fertility or investment behavior. Since most of the child's future outcomes are determined by the fixed endowment component, marginal investment plays a smaller role. However, as income rises, private resources become a larger part of what determines quality, and the classic Q–Q trade-off begins to shape behavior. Parents begin to allocate more income toward fewer children in order to enhance quality through direct investment. Under certain theoretical conditions, such as equal utility elasticities for quantity and quality, this framework produces a non-monotonic relationship between income and fertility. Fertility may decline as income rises at first, which reflects the desire to invest more intensively per child. However, beyond some point, once the marginal return to investment begins to flatten or saturate relative to the fixed endowment, fertility may increase again.

The U-shaped prediction emerges only under specific assumptions, and its validity depends on both the shape of the utility function and how endowments relate to parental background. More broadly, Becker and Tomes (1976) enriched the Q–Q framework by incorporating elements that reflect real-world variation. They argued that not all differences in child outcomes are the result of deliberate parental choice. Random factors, biological traits, and socioeconomic settings play a role. In this light, public policies, such as subsidized schooling, early childhood programs, or universal healthcare, can influence private fertility and investment decisions by shifting the effective value of E across the population. If government programs raise the floor for child endowments, then even low-income parents can achieve better outcomes without large private sacrifices. These policy-induced shifts in E alter the perceived return to having more children or investing more per child.

Becker and Tomes (1976) also considered the possibility that endowment is not randomly assigned but may vary systematically with income. Higher-income households may produce children with higher E due to better maternal nutrition, access to prenatal care, lower exposure to environmental risk, or assortative matching on traits associated with educational or occupational success. In these families, not only are the resources available for investment greater, but the potential gains from investment may also be higher, because children are better positioned to benefit from those inputs. This interaction deepens the divide between high- and low-income households, making it harder for disadvantaged families to catch up. As a result, inequality can persist or even widen across generations.

Finally, the model provides a mechanism for understanding how imperfections in credit markets can lead to persistent disadvantages. If parents with low income and low-E children cannot borrow to finance quality-enhancing investment, then the next generation begins life with the same disadvantage. Without external

intervention or structural change, this loop continues, which results in a pattern where poor families remain poor and rich families accumulate further advantage. The Becker-Tomes framework thus connects household-level decisions to bigger questions about the intergenerational transmission of human capital.

Parallel to Becker and Tomes's static analysis of endowments, another strand of the literature developed a fully dynamic version of the Q-Q model by incorporating parental altruism toward children's welfare. In this framework, introduced by Barro and Becker (1989), parents care not only about the number and quality of their children but also about the utility their descendants will enjoy in the future. Altruism in this context means that parents treat their children's utility as part of their own, thus extending the household's objective across generations. For example, a parent may reduce personal consumption to pay for a child's schooling, motivated not just by the child's immediate benefit but by the satisfaction the parent gains from the child's long-term success. This leads to a formulation of dynastic utility, where the household's objective spans infinitely many periods and takes the form of a recursive altruistic structure. A representative formulation is

$$U_0 = \sum_{t=0}^{\infty} \beta^t \, u(c_t, n_t),$$

where c_t denotes the consumption of the t-th generation, n_t the number of children, and $\beta \in (0, 1)$ the intertemporal discount factor. Given this structure, having an extra child n_t enters utility positively, but each child is assumed to receive the same utility as the parent if raised at a comparable standard of living. As a result, parents confront an intertemporal trade-off: having more children expands the number of future utility streams but also stretches current resources, since each child requires support. This trade-off gives rise to an Euler equation for optimal fertility choice, analogous to an optimal growth condition.

An implication of the dynastic model is that fertility decisions are sensitive to macroeconomic conditions, such as the interest rate or the rate of return on capital. A rise in interest rates increases the opportunity cost of channeling resources into children rather than saving, which tends to reduce current fertility—a substitution effect across generations. At the same time, higher returns make future generations wealthier, and this anticipated prosperity enters the utility calculations of parents in more complex ways. Barro and Becker (1989) demonstrated that the model can account for observed fertility responses to economic fluctuations and policy interventions It can also explain historical phenomena such as postwar baby booms and subsequent fertility declines through shifts in returns or labor-market opportunities.

In many dynastic models, child quality appears indirectly, often through the child's future human capital or income. One variant assumes parents value the aggregate human-capital stock of their children. This specification, combined with altruism, produces a similar trade-off: concentrating resources in fewer children raises each child's human capital, which raises the dynasty's long-run welfare.

These intergenerational extensions link micro-level fertility decisions to macroeconomic outcomes. By the late 1980s, work by Becker, Barro, and others had recast fertility as an endogenous choice that interacts with capital accumulation, income distribution, and policy. This laid the foundation for unified growth theories, which view the quantity-quality mechanism as central to demographic transition and long-run development.

Unified Growth Models

The unified growth theory, developed in the late 1990s and 2000s (notably by Oded Galor and co-authors), seeks to explain in one framework the entire sweep of economic development – from Malthusian stagnation, through the demographic transition, to modern growth. A central puzzle it addresses is why fertility rates, which were historically high and invariant to income in the Malthusian era, began to decline sharply in tandem with industrialization and rising incomes, eventually stabilizing at much lower levels in developed economies. The Q–Q trade-off provides a key part of the answer in these models. Galor and Weil (2000) and Galor and Moav (2002) explicitly incorporate parental choices about the quantity and quality of children and show how changes in the economic environment alter those choices and trigger demographic transitions.

In Galor and Weil (2000)'s model, for instance, technological progress gradually increases the return to human capital, especially in skilled occupations. In the early stages, when production relies on basic tools and techniques, unskilled labor holds more value. Under these conditions, parents have little reason to invest in formal schooling. Children are expected to contribute economically through agricultural work, domestic tasks, or low-skill jobs in workshops and factories. Fertility remains high because children impose a low financial burden and generate immediate returns. As technology becomes more advanced—such as during the Industrial Revolution—the earnings gap between skilled and unskilled labor widens. Education begins to offer significant advantages in the labor market. In response, parents adjust by having fewer children and placing greater emphasis on each child's development, including school attendance and better health care.

Evidently, industrialization raises the economic value of skilled labor, which alters household incentives. As returns to education increase, parents begin to favor investments in child quality over child quantity. This shift results in declining fertility because families choose to have fewer children and allocate more resources to each. The feedback effect is significant: higher educational investment raises productivity in the next generation, which in turn accelerates technological advancement and further increases the returns to human capital. Over time, the economy moves from a state of high fertility and low growth to one characterized by low fertility and sustained growth.

Furthermore, Galor and Moav (2002) introduced an evolutionary refinement to the unified growth framework by accounting for heterogeneity in parental preferences. During the Malthusian period, some families placed greater emphasis

on child quality, such as education, while others prioritized quantity. In a stagnant economy with limited returns to education, high-fertility lineages maintained a numerical advantage and suppressed average human capital. As technological progress increased the returns to education, families that valued quality gained an economic edge. Their children acquired more human capital and achieved higher income and survival rates. These advantages allowed such families to grow in relative size. Over time, this process resembled a form of evolutionary selection, gradually favoring quality-oriented parental types and shifting the population toward greater emphasis on child human capital. These dynamics strengthened the shift from high-fertility, low-education regimes to low-fertility, high-investment family structures. In formal overlapping-generations models, Galor and Moav (2002) demonstrate that this evolutionary adaptation accelerates the demographic transition. Their framework accounts for the rapid and widespread drop in fertility once it takes effect. Higher returns to human capital push parents to favor quality, while preferences for quality begin to dominate within the population. These forces support the emergence of a low-fertility, high-investment equilibrium and establish a unified explanation for both economic development and demographic change.

Importantly, unified growth models identify several complementary mechanisms that reinforce the basic Q–Q trade-off during development. One is the decline of child labor. As the economy modernizes, the value of child labor falls, both because legal reforms often restrict child labor and because parents realize the earnings their children could make as unskilled laborers are paltry compared to the potential returns if those children instead spend time in school. Hazan and Berdugo (2002) formally show that when child labor becomes less profitable relative to adult (skilled) labor, parents further reduce fertility and invest more in each child's education. Historical evidence from England, for instance, indicates that during industrialization the wages of children (relative to adults) dropped significantly, especially in skilled families, and this was accompanied by parents pulling children out of work to send them to school. Galor and Moav (2006) even argue that capitalist industrialists supported public education laws and child labor bans as a way to increase the human capital of the workforce, inadvertently hastening the fertility transition.

Another mechanism is the rise in life expectancy and child survival. Improvements in sanitation, nutrition, and medical knowledge in developing societies led to more children surviving to adulthood. While the earliest unified growth models treated mortality as exogenous or ignored it, later research demonstrated that declining child mortality can trigger lower fertility as well– parents no longer need "extra" births for insurance once they are confident their existing children will survive. In other words, increased child survival and the quality–quantity tradeoff are complementary explanations for fertility decline that operate in tandem. When fewer births are lost to disease, parents can achieve a desired number of surviving offspring with fewer total births, and they tend to reallocate effort into

each child's health and education.

The overall effect is a reinforcing cycle: better health raises the returns to schooling (healthier children can learn more effectively and have longer working lives), which further encourages educational investments and reduces fertility. Indeed, Galor notes that human capital should be interpreted broadly to include health as well as schooling; in unified growth theory, improvements in nutrition and physical well-being were crucial to making labor more productive and thus were part and parcel of the rise in demand for human capital.

The unified growth literature places the Q–Q model within a more general account of economic and demographic change. In this framework, higher income or stronger returns to child quality reduce fertility and help shift economies from stagnation toward sustained growth. Several mechanisms support this transition, such as a fall in child labor, a drop in child mortality, and a shift in parental priorities. These models explain not only the presence of a quantity-quality trade-off but also its rising influence at a specific point in history. The evidence supports these claims: countries that saw earlier increases in returns to education experienced earlier fertility decline, while delays in reforms, such as public education or health access, corresponded to prolonged high fertility. As a result, the Q–Q mechanism forms a key component of unified growth theory.

B. Recent Refinements to the Q-Q Model

Contemporary research has further refined the quantity-quality model by relaxing some of its initial simplifying assumptions. Three important extensions involve (1) capital market imperfections that constrain parents' ability to invest in child quality, (2) intra-household conflict and bargaining between mothers and fathers over fertility choices, and (3) recognition that child quality is multidimensional, which extends beyond schooling to include health, nutrition, and other facets of human capital.

To begin, I examine how credit constraints can give rise to poverty traps. The canonical quantity-quality (Q–Q) framework assumes that parents can reallocate resources freely; borrowing against future earnings to finance schooling or health investments whenever the expected return is high. In practice, especially in low-income settings, credit markets function imperfectly: poor households typically cannot secure loans to cover children's education or medical costs even when such investments would yield substantial future gains. This market failure magnifies the Q–Q trade-off. Becker, Lewis, and Willis–already noted by Grawe (2008) as emphasizing "resource limitations"–implicitly recognized this issue, but contemporary models make it explicit by imposing a borrowing constraint. Parents must fund childrearing from current income alone; they cannot collateralize a child's future wages to pay today's school fees. Consequently, when income is low, each additional birth directly reduces the attainable quality per child, potentially trapping families in a low-income–high-fertility equilibrium.

Formal models show that "the quality-quantity trade-off arises from a bind-

ing credit constraint that prevents parents from borrowing against future child income." Empirical work supports this mechanism. Kremer and Chen (2002) and De La Croix and Doepke (2003) document that countries facing tighter liquidity constraints tend to display higher fertility and lower educational attainment, consistent with liquidity-constrained parents favoring quantity over quality. Cross-country evidence likewise indicates that where financial frictions are more severe, the negative correlation between fertility and schooling is stronger. Theoretically, introducing a borrowing limit can generate multiple steady states: one with low fertility and high investment when incomes suffice to cover quality costs, and another with high fertility and minimal investment when they do not. Policy instruments such as education subsidies or conditional cash transfers effectively relax these constraints, which nudge households toward the low-fertility, high-investment equilibrium. In short, incorporating credit market imperfections deepens the explanatory power of the Q-Q model: economic growth alone may not reduce fertility if households remain too cash-poor to afford schooling, whereas targeted quality-enhancing transfers can catalyze both demographic and human-capital transitions.

A further refinement of the quantity-quality (Q–Q) model considers the question of *who* within the household makes fertility and child investment decisions. The original Beckerian framework adopts a unitary model of the family, and assumes a single utility function and complete agreement between spouses over optimal fertility n and child quality q. In practice, however, empirical evidence reveals significant heterogeneity in preferences between household members along gender lines. According to Oppenheim Mason (1987); Thomas (1990), for instance, men often desire more children than women and may differ in their willingness to invest in each child's education or health. These discrepancies have motivated game-theoretic models of intra-household bargaining, in which fertility and investment outcomes reflect the relative influence of each parent's preferences.

In such models, the mother is typically assumed to have a stronger preference for child quality—such as health and schooling—while the father may favor either more children or alternative uses of household resources. The resolution of these conflicting preferences depends on bargaining power, which can be shaped by income contributions, legal rights, cultural norms, or access to external resources. When the mother's bargaining power increases, theoretical models predict a shift toward lower fertility and higher per-child investment, holding other factors constant. This prediction is consistent with empirical findings: Iyigun and Walsh (2007); Doepke and Kindermann (2019) show that greater female empowerment—via education or labor force participation—correlates with reduced fertility and increased investment in child human capital.

Mathematically, the household's first-order condition for fertility in a bargaining model can be written as:

$$\alpha \frac{\partial U_{\text{wife}}}{\partial n} + (1-\alpha)\frac{\partial U_{\text{husband}}}{\partial n} = \lambda(p_n + p_q q),$$

with a corresponding condition for q. Here, $\alpha \in [0, 1]$ represents the wife's bargaining weight. When α increases, the composite marginal utility of additional children typically decreases—especially if the wife prefers fewer children—leading to lower equilibrium fertility and a shift along the Q–Q frontier toward higher quality.

Recent work also explores dynamic bargaining, in which spouses negotiate sequential decisions over time, potentially leading to strategic behavior (e.g., one partner may accelerate or delay subsequent births). Although these models introduce complexity, their core implication for the Q–Q framework is clear: household power dynamics fundamentally shape the trade-off between child quantity and quality. In societies where women have limited decision-making autonomy—due to lack of access to contraception, or social norms—fertility tends to remain high and per-child investment low, which stalls demographic transition. Conversely, when women gain bargaining power—through legal reforms, labor market participation, or targeted transfers—the household often reallocates resources toward fewer but higher-quality children.

This theoretical insight is corroborated by policy experiments. For instance, cash transfer programs directed to mothers consistently lead to greater spending on children's health, education, and nutrition compared to equivalent transfers given to fathers (Duflo, 2003; Thomas, 1990) . Such outcomes support the hypothesis that mothers place higher weight on child quality, and that who controls the purse strings matters deeply. In sum, introducing intra-household bargaining into the Q–Q model enriches its explanatory scope: it highlights how family structure and power asymmetries—not just income levels or prices—generate variation in fertility and human capital outcomes across and within societies.

C. Health and Nutrition in the Q–Q Model

The original Q–Q models often used a single catch-all variable for child quality, typically thought of as education or "expenditure per child." Recent work emphasizes that child quality is multi-faceted, and that parents make trade-offs along several dimensions of investment—cognitive development, health, nutrition, etc. This is salient in developing countries, where basic health and nutrition are pressing concerns alongside schooling.

The theoretical question is how these dimensions interact with the quantity decision. If parents allocate a budget across, say, schooling q_{edu} and nutrition/health q_{health} for each child, then having more children forces cutbacks in both dimensions (unless parents reallocate across them). In some models, health and education are complementary: a healthier child benefits more from education, and an educated mother might raise a healthier child. This complementarity can amplify the Q–Q trade-off—investing in one dimension (health) increases the returns to investing in the other (education), so a high-quality strategy becomes more focused on fewer children.

On the other hand, if one dimension has diminishing returns more quickly

than another, parents might prioritize achieving a threshold level of health for all children before adding more education, which creates a nonlinear effect on fertility. One especially important aspect of health in the Q–Q framework is child survival. The probability that a child survives to adulthood effectively multiplies the utility of having that child. Historically, high child mortality led to a strategy of "quantity for insurance," where parents had additional births to ensure survivorship. As mortality falls due to public health improvements, parents can shift toward quality without risking childlessness (Kalemli-Ozcan, 2002; Kalemli-Ozcan, Ryder and Weil, 2000).

Kalemli-Ozcan (2002) developed a stochastic model in which fertility choices are made under uncertainty about child survival. Her results show that declining mortality causally reduces fertility and increases educational investment per child, in line with the Q–Q trade-off. In unified growth models, declining child mortality reinforces the demand-for-human-capital channel of the demographic transition (Galor and Moav, 2004).

Beyond survival, early-life health indicators such as birth weight and vaccination status are important dimensions of child quality, especially in low-income settings. When resources are limited, parents may struggle to meet the basic needs of each child. Empirical evidence aligns with the quantity–quality framework: children from larger families often have worse health outcomes at birth and are less likely to receive core immunizations. For example, recent multi-country data show that the share of completely unimmunized children—so-called "zero-dose" children—increases from 10.5% among those with no siblings to 17.2% among those with four or more (Costa et al., 2024). Studies from Southeast Asia also report that higher birth order correlates with lower average birth weight. These patterns suggest that as fertility increases, the health of each child may suffer due to more limited parental investment. Studies from Southeast Asia (Chen, 2021) also report that higher birth order correlates with lower average birth weight.

Similar patterns emerge in sub-Saharan Africa (Bishwakarma and Villa, 2019) and South Asia, where first-born children experience slower growth once younger siblings arrive—again pointing to intrahousehold trade-offs. Theoretically, one can extend the Becker model with a nutrition production function, where a child's health H depends on food F and medical care M, such that:

$$H = g(F, M)$$

and H enters either utility directly or the child's future productivity. Parents then choose n, F, M, and possibly schooling per child. Larger n reduces F and M per child, lowering H; if parents place high value on H, or if H enhances returns to education, they will opt for smaller families.

Importantly, improvements in nutrition (through income growth or public programs) can first lead to both better health and higher fertility (since healthier women can bear more children). But over time, as standards of living rise, better nutrition increases the returns to investing in fewer, healthier children. Economic

historian Fogel (1994) argues that Europe's demographic transition was partly driven by better diets: improved nutrition raised the productivity of educated workers, which made education more valuable and shifted family preferences toward quality.

These propositions are important for modern development policy. Family planning programs that encourage lower fertility without improving child health and education may have limited long-term effects. Conversely, health and nutrition interventions—such as vaccinations or food supplementation—can enhance the returns to education and induce parents to reduce fertility voluntarily. The extended Q–Q framework thus serves as a unifying tool to understand how diverse interventions—ranging from school fees to nutrition programs—shape long-run development trajectories.

D. Philippine Evidence on the Q–Q Trade-off

Early empirical work in the Philippines provided suggestive support for this theory. For example, an influential study by Horton (1986) used Philippine household data to jointly examine fertility and child nutrition and treated nutritional status as a measure of child quality. Horton found that better-educated mothers and fathers tended to choose smaller families and achieved better-nourished children–evidence of substitution away from "quantity" toward child "quality." Notably, she observed strong birth-order effects on nutrition (first-born children faring better than later-born), which hint that parents may not evenly distribute quality investments among all children. This early work indicated a quantity-quality (Q–Q) trade-off in Philippine families, though it largely documented correlations rather than definitive causation.

Subsequent studies in the Philippines have tackled the critical issue of causality, using innovative research designs to isolate exogenous changes in family size. One seminal contribution is Orbeta Jr (2010), who examined the impact of family size on children's schooling using a nationally representative survey. Recognizing that family size is endogenous (parents' fertility choices may reflect unobserved preferences or constraints), Orbeta employed an instrumental-variable (IV) approach grounded in Becker's framework. Specifically, he used the sex composition of the first two children as an instrument for having additional children – an approach pioneered by Angrist and Evans (1996) – leveraging the fact that Filipino parents often desire a mixed-gender sibset (and are more likely to have a third child if the first two are the same sex) (Vicerra and Cruz, 2013). This strategy aims to generate random-like variation in family size uncorrelated with parental characteristics.

The IV estimates confirmed a significant negative causal effect of higher fertility on educational outcomes. Orbeta Jr (2010) found that each additional child in the household reduced the proportion of school-age children (6–24 years) attending school by roughly 19% of the baseline attendance rate. The trade-off was especially pronounced at higher education levels: for example, the estimated drop

in school attendance was about 26% at the secondary level and 57% at the tertiary level for each additional sibling. These are sizable effects, implying that children from large families are substantially less likely to remain in school, presumably due to tighter household budget constraints or diluted parental attention. Moreover, the burden of the trade-off appeared regressive: Orbeta's results showed much larger schooling deficits from an extra child in poorer households than in richer ones. For instance, in the poorest quintile, an additional sibling reduced school attendance by an estimated 24% (for ages 6–24), compared to a 16% reduction in the richest quintile. This regressive pattern aligns with Becker's theory that resource constraints bind more tightly for low-income families, which makes the Q–Q trade-off more acute. In summary, Orbeta's study – the first in the Philippines to account for fertility endogeneity – provides robust evidence that increases in family size cause significant declines in child educational attainment and validates the quantity-quality trade-off in this context. Large family size thus emerges as one mechanism contributing to poverty, by impeding children's human capital accumulation in the Philippines.

Further compelling evidence comes from a natural experiment studied by Dumas and Lefranc (2019). They exploit a unique policy shock in metropolitan Manila to identify the trade-off. In 1998, the Mayor of Manila city imposed a sudden ban on modern contraceptives in public facilities, drastically curtailing access to family planning for residents of Manila city (but not in surrounding municipalities). Dumas and Lefranc use this policy as a quasi-experiment: comparing families in Manila city (treated by the ban) to similar families in other cities unaffected by the ban before and after 1998. This difference-in-differences design, coupled with the fact that older mothers were naturally less fecund during the ban, isolates an exogenous fertility increase.

The results are striking. The contraceptives ban led to a significant rise in births and family size in Manila city relative to the control areas. Correspondingly, children born in Manila during the ban era experienced a sizable decline in educational attainment – clear evidence of a Q–Q trade-off precipitated by the shock. In the authors' words, the policy-driven increase in family size "provide[s] evidence of a quality–quantity trade-off": larger families, forced by the ban, resulted in lower schooling outcomes per child.

Beyond education, researchers have also examined health and other child outcomes in relation to family size. Evidence generally suggests the Q–Q trade-off extends to child health and nutrition. For instance, Horton (1986) already hinted that large families may compromise child nutrition for later-born siblings. More recent regional research resonates with this. Hatton et al. (2018) analyze the effect of fertility on child height (a long-run health indicator) using longitudinal data from Indonesia – a neighboring Southeast Asian country with comparable developmental challenges. They address endogeneity by exploiting Indonesia's family planning program rollout and exposure to mass media as instruments for fertility. The authors find a significant negative impact of family size on child health: each

additional sibling is associated with about a one-third standard deviation reduction in a child's height-for-age Z-score, after controlling for other factors. This health penalty from having more children is strongest in low-education households and appears in both urban and rural areas. Such findings mirror the Philippine evidence that the harms of large family size are most pronounced among disadvantaged families. In economic terms, poorer parents with many children struggle to provide adequate nutrition and schooling to all, which highlight the equity dimension of the Q–Q trade-off.

E. Family Size and Child Outcomes in Southeast Asia

The Philippine experience is echoed in other ASEAN countries, where researchers have probed the quantity-quality trade-off with diverse outcomes and methods. In Vietnam, for example, rapid fertility decline alongside rising education led to questions about a Q–Q mechanism. Anh et al. (1998) documented a negative correlation between family size and children's school enrollment in Vietnam, though their analysis could not fully establish causality.

More rigorously, Dang and Rogers (2016) used distance to the nearest family planning center as an instrument to study Vietnamese households' investments in education. They introduced a novel measure of child quality—spending on private tutoring, a prevalent form of educational investment in Vietnam—alongside traditional indicators like schooling expenses. The IV estimates confirmed that children with more siblings receive significantly lower educational investments from their families. In particular, Vietnamese families of larger size spent less on each school-age child's schooling and tutoring, even after controlling for community factors. This effect was robust across different definitions of family size and model specifications, indicating that Vietnamese parents do indeed trade off quantity for quality when faced with resource constraints. Such evidence aligns squarely with Becker's model: as Vietnamese family size increases, per-child education spending falls; parents are prioritizing "quality" less when they have more offspring.

Indonesia and Thailand show similar patterns. Demographic research in Thailand during its fertility transition found that large families had markedly worse educational outcomes. Knodel, Havanon and Sittitrai (1990), studying Thai data in the 1990s, observed that once family size exceeded about 4–5 children, the likelihood of a child progressing to or staying in secondary school dropped precipitously compared to smaller families. Although these early Thai studies were based on correlations, they strongly suggested that limited family resources were being spread thin in big families and hurt children's schooling attainment.

In Indonesia, cohort analyses and natural experiments reinforce the trade-off. Maralani (2008) showed that the relationship between sibship size and schooling evolved from neutral or even positive for older cohorts (born when education opportunities were limited) to negative for more recent cohorts, consistent with a growing importance of education in a modernizing economy. More concretely,

the aforementioned study by Hatton et al. (2018) in Indonesia provides causal evidence that mirrors the Philippine findings in health and nutrition. Likewise, an analysis of Indonesian census data (Feng, 2021) found that having additional siblings significantly lowers children's educational attainment once birth order effects are accounted for, paralleling results from China and Vietnam.

These regional studies share a commonality: in resource-constrained settings of Southeast Asia, increased child quantity tends to come at the expense of child quality, be it years of schooling, academic spending, or health status. The consistency of this pattern—across countries with different cultures and policies—highlights the fundamental economic logic identified by Becker and Lewis (1973). Parents with finite resources face difficult choices, and many appear to balance quantity and quality in a way that confirms the trade-off hypothesis.

Despite these similarities, there are some noteworthy nuances and gaps in the ASEAN literature. One is the role of public policy and development level. Evidence suggests that the Q–Q trade-off may be mitigated in contexts with strong public support for education and health. For instance, studies in developed countries (e.g. Israel, Norway) often find little or no trade-off once factors like birth order are accounted for (Black, Devereux and Salvanes, 2005; Kristensen and Bjerkedal, 2010; Angrist, Lavy and Schlosser, 2010). In Southeast Asia, however, public education quality and social safety nets are still developing, and the cost of raising children (education fees, food, etc.) is largely borne by families themselves (OECD, 2024). This may explain why the trade-off emerges so clearly in the Philippines, Vietnam, Indonesia, and Thailand.

Another nuance is methodological: more recent studies employ credible identification strategies (IVs, twins, policy shocks) and consistently find a causal negative effect of family size on child outcomes, whereas older studies without such controls sometimes found weaker effects or none at all. This highlights the importance of accounting for endogeneity.

Finally, there remain gaps for future research. Most ASEAN studies focus on education and early-life health indicators as measures of child quality; there is relatively little evidence on long-term outcomes such as children's eventual earnings or income in adulthood. It is not yet fully clear whether the schooling and health disadvantages observed in larger families translate into significantly lower adult productivity or income—a link that Becker and Tomes (1976) theorized but which could be explored further in this region.

Additionally, while the trade-off appears pervasive, its magnitude can vary: for example, the penalty of an extra child may be larger in poorer rural areas than in urban or wealthier settings, suggesting that local context (poverty, gender norms, access to services) can modulate the trade-off. Comparative studies across ASEAN are still somewhat limited, and a common challenge is disentangling related factors like birth order, sibling composition, and parental preferences.

Nonetheless, the prevailing evidence from the Philippines and its regional neighbors strongly supports Becker's quantity-quality conjecture. As families

have fewer children, they appear to invest more in each child's education—investments crucial for human capital development and economic growth. Conversely, high-fertility households risk under-investing per child, which reinforces cycles of poverty and inequality. This literature thus provides an important empirical foundation for policies in the Philippines and ASEAN—from family planning programs to education subsidies—that aim to ease the quantity-quality trade-off and help families achieve both manageable size and better outcomes for the next generation.

III. Conceptual Framework and Hypotheses

This study builds upon the foundational framework of the quantity-quality (Q–Q) trade-off, first articulated in Becker (1960) and later refined in Becker and Lewis (1973). In this model, fertility and child quality are jointly determined under a full-income constraint, such that increases in the number of children—without a corresponding expansion in household resources—necessitate a reduction in the average investment per child. While empirical applications of this framework have traditionally focused on educational outcomes, child quality can also be expressed in terms of early-life health, including indicators that emerge at birth or shortly after, such as birth weight, and uptake of essential vaccinations.

This analysis focuses specifically on early-life health as a proxy for child quality. These outcomes, unlike cognitive or schooling-related investments, are biologically constrained and occur within a narrow developmental window, during which parental inputs are least substitutable and most consequential. The trade-off is expected to be especially salient in settings where fertility increases exogenously while family resources remain fixed. In such contexts, a sudden increase in sibship size may compel households to spread food, healthcare, and caregiving attention across more dependents, which would potentially lead to diminished health investments and outcomes for each child.

To formalize this relationship, consider a household with total income I, choosing nutritional investments κ_j for each of n children, along with consumption Z unrelated to child quality:

$$I = \sum_{j=1}^{n} \kappa_j + Z.$$

In the presence of a fertility shock, holding I constant, the average κ_j must fall unless offset by changes in parental behavior or external transfers. If these inputs decline and are critical to early-life development, observable deficits may arise in birth weight, and children may be less likely to receive complete immunization schedules.

The first hypothesis derived from this framework is that an exogenous increase in fertility leads to a decline in per-child health investment, particularly in low-income urban households with little financial slack. A second, related hypothesis

posits that this decline results in measurable early-life health disadvantages—specifically, lower birth weight and reduced uptake of key childhood vaccinations. These hypotheses describe a causal pathway through which fertility shocks may contribute to early deprivation and potentially reinforce intergenerational cycles of poor health and limited human capital.

IV. Data and Empirical Strategy

A. Data

This study draws on two primary data sources: the Philippine Demographic and Health Surveys (DHS) and the Integrated Public Use Microdata Series (IPUMS) census data. The DHS provides detailed child- and household-level information on fertility behavior, maternal characteristics, and early childhood health outcomes. I use three rounds of the DHS—1998, 2003, and 2008—which are the only Philippine DHS waves with publicly available GPS cluster data. These data are used to classify respondents into treatment and control groups. Because city-level identifiers are not included in the public-use DHS files, I rely on approximate geographic assignment using cluster coordinates. Clusters located within or near the administrative boundaries of the City of Manila are classified as treated, while clusters located elsewhere in the National Capital Region (NCR) serve as the control group.

The DHS analytic sample is limited to children under five years of age who were living with their mothers at the time of the survey. I examine three core early-life health outcomes: (1) reported birth weight (in grams); (2) maternal perception of birth size; and (3) vaccination uptake, measured as the receipt of key early childhood immunizations (BCG, DPT, polio, and measles). These outcomes are consistently available across all three survey rounds and are commonly used as proxies for child quality in studies of the quantity–quality trade-off.

To complement the DHS analysis and support the identification strategy, I construct a panel of individual-level microdata from the IPUMS harmonized Philippine censuses (1990, 2000, and 2010). These nationally representative surveys provide a snapshot of women of reproductive age (15–49) and contain detailed fertility histories—including total children ever born, births in the past year, and number of surviving children. I limit the sample to women ages 15–49 and construct a composite fertility index combining several measures: total children ever born, births in the past year, number of children under age five, and the age of the youngest child. By comparing Manila with other provinces and municipalities in NCR, I test whether long-run fertility patterns reflect the contraceptive policy shock.

The 1998 DHS serves as the primary pre-policy baseline, capturing births prior to the issuance of Executive Order No. 003. The 2008 DHS is the main post-policy round, while the 2003 DHS is treated as a transition period. I exclude the 1993 DHS due to the absence of geospatial identifiers. In contrast, the census years

1990 and 2000 bracket the policy's onset, while the 2010 census provides post-policy fertility outcomes. This combination of sources enables both short-run and long-run assessments of the policy's impact on fertility and child investment.

To validate the policy shock, I use DHS region- and city-level contraceptive use indicators, which confirm a sharp and sustained decline in modern contraceptive use in Manila relative to other NCR cities. These patterns reinforce the plausibility of Executive Order No. 003 as an exogenous shock to fertility preferences and access. I also considered the Philippine National Nutrition Survey (NNS) as an auxiliary source, but its limited pre-2003 coverage restricts its utility for identifying pre-policy trends. For this reason, the combined DHS and IPUMS datasets offer the most comprehensive and temporally balanced data available for the study's objectives.

B. Variables

The primary outcome variables reflect early-life health and investment. These include: (1) birth weight in grams, as recorded by the mother when available; and (2) indicators for whether the child received core early-life vaccinations, including BCG, DPT1–3, polio doses, and measles.

The main explanatory variable is sibship size, defined as the total number of surviving children ever born to the mother at the time of the child's birth, as reported in the birth history module. This variable captures the resource dilution mechanism central to the quantity–quality trade-off. To isolate exogenous variation in sibship size, I construct an instrument for fertility based on policy exposure: an interaction of a Manila indicator and a post-ban cohort indicator. Specifically, children born in the City of Manila in or after 2000 are coded as treated. In extended specifications, I interact this treatment indicator with maternal age at birth to allow for heterogeneity in fertility responses across age cohorts, considering the fact that older women likely exhibited lower responsiveness due to reduced fecundity.

Control variables include standard child-, maternal-, and household-level characteristics. At the child level, I control for sex, birth order, multiple birth status, and the preceding birth interval in months. Maternal covariates include age at birth, educational attainment (none, primary, secondary, higher), literacy, employment status, and age at first birth. Household-level controls include household size, urban/rural residence (restricted to NCR in the main analysis), and the DHS wealth index. All regressions include survey year fixed effects to control for secular trends and common shocks. Estimates are weighted using DHS sample weights, and standard errors are clustered at the primary sampling unit (PSU) level.

C. Estimation

To estimate the causal effect of fertility on early-life health outcomes, I implement a two-stage least squares (2SLS) strategy. The first stage is estimated at the

maternal level, as fertility is a cumulative outcome of the mother rather than the child. Importantly, this stage also serves to establish whether the contraceptive supply ban had a measurable impact on maternal fertility behavior. Specifically, I use two measures of fertility: the total number of children ever born and the number of living children at the time of survey. These outcomes capture distinct dimensions of fertility—cumulative births and net surviving offspring—and are regressed on an indicator for policy exposure, defined as the interaction between a Manila municipality indicator and a post-ban birth cohort.

In the second stage, I shift to the child level and use the number of surviving older siblings at the time of the index child's birth as the endogenous regressor, instrumented by policy exposure. This variable captures sibship size at birth, and allows me to estimate the effect of increased family size—induced by the contraception ban—on early-life health outcomes such as birth weight, and number of vaccines received. This two-stage framework allows me to isolate plausibly exogenous variation in family size induced by the contraceptive supply ban in Manila, while addressing endogeneity concerns that arise when fertility is influenced by unobserved preferences or constraints. I estimate all models using linear probability models (LPMs) for consistency with the 2SLS framework. All specifications apply DHS sampling weights and cluster standard errors at the PSU level.

V. Results and Discussion

A. Descriptive Statistics

The following tables present summary statistics for key child health outcomes, disaggregated by location and policy period. Table 1 presents summary statistics for mean birth weight by location and policy period.

Table 1—Birth Weight by Location and Policy Period

Group	Birth weight (g)
Manila, Pre-Policy	3023.12 (712.62), n = 156
Manila, Post-Policy	2969.79 (619.23), n = 144
Other NCR Cities, Pre-Policy	2830.41 (743.57), n = 70
Other NCR Cities, Post-Policy	2965.89 (793.72), n = 88
Rest of the Philippines, Pre-Policy	3106.41 (726.91), n = 4562
Rest of the Philippines, Post-Policy	2994.45 (681.71), n = 2298

Among children born in Manila, the average birth weight declined from 3,023.1 grams (SD = 712.6) in the pre-policy period to 2,969.8 grams (SD = 619.2) after the policy's implementation. A similar decline is observed in non-National Capital

Region (NCR) areas, where mean birth weight dropped from 3,106.4 grams (SD = 726.9) to 2,994.5 grams (SD = 681.7). In contrast, birth weights in other NCR cities remained relatively stable over time, increasing slightly from 2,830.4 grams (SD = 743.6) to 2,965.9 grams (SD = 793.7). The majority of births in the sample occurred in non-NCR areas, which is expected given that these include the rest of the country beyond Metro Manila. Notably, the decline in birth weight was more pronounced in Manila compared to other cities within the National Capital Region (NCR), suggesting a localized impact of the policy. While non-NCR areas also experienced reductions, the contrast between Manila and its neighboring NCR cities—where the policy was not implemented—is a more relevant comparison for isolating potential policy effects.

Lastly, Table 3 presents the distribution of childhood vaccine uptake across policy periods and geographic areas.

Table 2—Number of Vaccines Received by Location and Policy Period

Group	1–2 Vaccines	3–4 Vaccines	5+ Vaccines	No Vaccines
Manila, Pre-Policy	1 (0.5%)	4 (1.9%)	38 (18.2%)	166 (79.4%)
Manila, Post-Policy	3 (1.8%)	5 (3.0%)	33 (20.0%)	124 (75.2%)
Other NCR Cities, Pre-Policy	1 (0.8%)	3 (2.4%)	31 (25.2%)	88 (71.5%)
Other NCR Cities, Post-Policy	3 (2.2%)	5 (3.7%)	52 (38.2%)	76 (55.9%)
Rest of the Philippines, Pre-Policy	68 (0.9%)	136 (1.8%)	1869 (25.1%)	5364 (72.1%)
Rest of the Philippines, Post-Policy	95 (2.8%)	117 (3.4%)	1141 (33.5%)	2057 (60.3%)

Table 3 presents the distribution of childhood vaccine uptake across policy periods and geographic areas. A substantial share of children in all groups received no vaccines, especially in Manila (79.4% pre-policy, 75.2% post-policy). However, there was a notable post-policy increase in children receiving 5 or more vaccines in both NCR (from 25.2% to 38.2%) and non-NCR cities (from 25.1% to 33.5%). These shifts indicate modest improvements in vaccine coverage in areas outside Manila, which—being unaffected by the contraceptive access restrictions—merit further investigation into other potential drivers, such as enhancements in health system capacity or increased accessibility of immunization services.

Still, while the descriptive patterns suggest a localized effect in Manila—especially relative to nearby NCR cities—I use the full Philippine sample for the regressions that follow. This decision is mostly practical: restricting the analysis to NCR alone would leave too small a sample to detect meaningful effects with confidence. That said, I recognize the importance of comparing Manila to similar, unaffected areas. For robustness, I replicate all key models using only births from NCR and report those results in the Appendix.

B. First-Stage Estimates: Effect of the Contraceptive Ban on Fertility

To assess the fertility effects of Executive Order No. 003 (EO 003)—a 2000 Manila City ordinance that curtailed access to modern contraceptives—I estimate first-stage ordinary least squares (OLS) regressions that relate policy exposure to maternal fertility outcomes, using data from the Demographic and Health Surveys (DHS). These first-stage estimates serve as the foundation for the instrumental variable strategy in the next stage of analysis. The regressions use the mother as the unit of analysis, since fertility is a cumulative outcome determined at the maternal level. Evidence that the policy affected maternal fertility behavior provides a necessary condition for interpreting later estimates of child health outcomes as causal effects of fertility.

The regression specification is designed to isolate the differential change in fertility among mothers residing in Manila following the implementation of EO 003, relative to mothers in other regions over the same period. I estimate the following equation:

$$\text{Fertility}_{im} = \beta_0 + \beta_1 \text{Manila}_m + \beta_2 \text{ManilaPost}_{im} + \boldsymbol{X}'_{im}\gamma + \lambda_{y(i)} + \varepsilon_{im}$$

Where: $Fertility_{im}$ is the fertility outcome for mother i in municipality m (e.g., number of living children at survey, total children ever born); Manila_m is an indicator for residence in Manila,ManilaPost_{im} is an interaction term equal to 1 if the mother resided in Manila and gave birth in or after the year 2000 (the policy period); X_{im} includes maternal characteristics such as age, educational attainment, and household wealth; $\lambda_{y(i)}$ are birth year fixed effects, controlling for national trends in fertility by cohort, and ε_{im} is the error term, clustered at the primary sampling unit (PSU) level.

The regression results, shown in Table 4, examine the relationship between policy exposure and sibship size. I use two measures of maternal fertility: the number of living children at the time of survey (Model 1) and the total number of children ever born (Model 2). In both specifications, the coefficient on the interaction term Manila × 2000 is positive—0.098 in Model 1 and 0.206 in Model 2—though neither is statistically significant. These estimates suggest a directionally consistent increase in fertility among Manila women exposed to the contraceptive ban. Maternal education and household wealth are negatively associated with fertility, while maternal age is positively associated, consistent with socioeconomic gradients in childbearing.

Table 3 presents first-stage regression estimates assessing the relationship between exposure to Manila's post-2000 fertility-restrictive policy and sibship size. The outcome variables are the number of living children at the time of survey (Model 1) and the total number of children ever born, including deceased (Model 2). In both models, the interaction term Manila × Post-2000 is positive—0.098 (SE = 0.691) in Model 1 and 0.200 (SE = 0.461) in Model 2—indicating that

children born to Manila mothers after the policy may have experienced slightly larger sibship sizes, on average. Although these estimates are not statistically significant, their consistent positive direction suggests that the policy did not produce a strong fertility-decreasing effect, and may even have been associated with modest increases in completed or surviving fertility.

Table 3—First-Stage Regressions: Effect of Policy Exposure on Sibship Size

	Model (1)	Model (2)
Manila	0.158	0.242
	(0.333)	(0.142)
Manila × Post-2000	0.098	0.200
	(0.691)	(0.461)
Mother's Education	−0.326***	−0.397***
	(<0.001)	(<0.001)
Mother's Age	0.200***	0.221***
	(<0.001)	(<0.001)
Wealth Index	−0.250***	−0.291***
	(<0.001)	(<0.001)

+ p < 0.1, * p < 0.05, ** p < 0.01, *** p < 0.001
All regressions include birth year fixed effects.
Standard errors are clustered at the PSU level.
Estimates are weighted using DHS sample weights.

The control variables function as expected and lend credibility to the model. Maternal education is significantly associated with smaller sibship sizes (−0.326 and −0.397, $p < 0.001$), while maternal age is positively associated (0.200 and 0.221, $p < 0.001$), reflecting life-cycle fertility dynamics. Wealth index is also negatively associated with fertility (−0.250 and −0.291, $p < 0.001$). Both models explain substantial variation in the outcome, with adjusted R^2 values of 0.503 and 0.527, respectively.

Meanwhile, Table 4 serves as a placebo test using Quezon City[1], an urban area demographically and geographically comparable to Manila but not subject to the policy. The placebo interaction term Quezon × Post-2000 is near zero in both specifications: 0.006 (SE = 0.951) for living children and 0.036 (SE =

[1]Quezon City and Manila are both highly urbanized cities within Metro Manila and share closely aligned demographic profiles. Data from the 2010 Census and the 2013 NDHS show that both cities had similar rates of secondary or higher education among women (approximately 55–60%), high institutional delivery coverage (above 90%), and comparable access to modern contraceptive methods. Prior to the policy, total fertility rates in both cities ranged between 2.5 and 2.7 children per woman. These commonalities extend to urban infrastructure and wealth distribution, and make Quezon City a credible counterfactual for isolating the effects of Manila's 2000 policy.Philippine Statistics Authority (2012); National Statistics Office and Macro International Inc. (1999); Philippine Statistics Authority and ICF International (2014); Metropolitan Manila Development Authority (2012)

0.754) for total children ever born. These findings are precisely null and much smaller in magnitude than those observed for Manila, which provide important reassurance that the patterns seen in Table 3 suggest that the observed effects are not confounded by region-wide demographic shifts or by secular trends in urban fertility behavior. In contrast to Manila, there is no directional indication of post-2000 fertility increase in Quezon City.

Table 4—Placebo Regressions: Using Quezon City as Falsification Test

	Model (1)	Model (2)
Quezon City	0.025	0.011
	(0.750)	(0.904)
Quezon × Post-2000	0.006	0.036
	(0.951)	(0.754)
Mother's Education	−0.325***	−0.396***
	(<0.001)	(<0.001)
Mother's Age	0.200***	0.220***
	(<0.001)	(<0.001)
Wealth Index	−0.249***	−0.289***
	(<0.001)	(<0.001)

+ p < 0.1, * p < 0.05, ** p < 0.01, *** p < 0.001
Placebo test using Quezon City as a comparison group.
All regressions include birth year fixed effects.
Standard errors are clustered at the PSU level.
Estimates are weighted using DHS sample weights.

Table 5 strengthens this interpretation by directly comparing Manila to Quezon City in a difference-in-differences framework. Restricting the sample to these two cities, the coefficient on the Manila × Post-2000 term remains positive: 0.025 (SE = 0.925) in Model 1 and 0.104 (SE = 0.724) in Model 2. Although these estimates are again statistically insignificant, they are directionally aligned with the first-stage results and notably larger than those in the placebo regressions. This suggests that even when directly comparing policy-exposed and unexposed cities within a single model, the post-2000 trend in Manila is somewhat more pronounced, and offers support for a modest, policy-related fertility shift.

To complement the regression analysis, I construct approximate birth rates using four waves of the Philippine Census (1990, 1995, 2000, and 2010). I calculate city-level birth rates by year and compare trends across Manila, other cities in the National Capital Region (NCR), and the rest of the Philippines. Although these census-based estimates are less precise than those derived from the DHS, they provide an independent view of fertility patterns during the policy period. I

Table 5—Difference-in-Differences: Manila vs. Quezon City, Post-2000

	Model (1)	Model (2)
Manila	0.116	0.204
	(0.499)	(0.254)
Manila × Post-2000	0.025	0.104
	(0.925)	(0.724)
Mother's Education	−0.274***	−0.331***
	(<0.001)	(<0.001)
Mother's Age	0.156***	0.172***
	(<0.001)	(<0.001)
Wealth Index	−0.269***	−0.350***
	(<0.001)	(<0.001)

+ p < 0.1, * p < 0.05, ** p < 0.01, *** p < 0.001
Sample restricted to Manila and Quezon City.
All regressions include birth year fixed effects.
Standard errors are clustered at the PSU level.
Estimates are weighted using DHS sample weights.

estimate annual birth rates by dividing the number of young children (ages 0–5) in a given census year by the number of women of childbearing age who could have plausibly given birth to them, based on age and co-residence within the same household. To improve the likelihood of correctly linking mothers and children, I restrict the sample to women identified as household heads or spouses, under the assumption that children typically reside with their biological mothers.

Formally, for each calendar year t and maternal age group a, the approximate birth rate is defined as:

$$\text{BirthRate}_{a,t} = \frac{\text{Number of children aged } c-t \text{ in census year } c}{\text{Number of women aged } c-t+a \text{ in census year } c}$$

This approach estimates the share of women aged a in year t who gave birth, inferred from the presence of children born in year t (that is, children aged $c-t$) in the census conducted in year c. I use census years $c \in 1990, 1995, 2000, 2010$ and construct birth rates based on children aged 0 to 5 at the time of enumeration, thereby covering calendar years from 1985 to 2015. To reduce noise and ensure comparability across time, I aggregate across maternal ages 15 to 49.

Table 6 compares average birth rates before and after 2000 across three region groups: Manila, other NCR cities, and the rest of the Philippines. The results reveal a notably smaller decline in birth rates in Manila relative to the comparison

areas. Between the pre-2000 and post-2000 periods, Manila's average birth rate decreased by only 8.4% (from 0.0822 to 0.0753). In contrast, other NCR cities experienced a 17.0% decline, and the rest of the country saw a 20.8% drop.

Table 6—Average Birth Rates Before and After 2000 by Region Group

Region Group	Pre-2000	Post-2000	Percent Change
Manila	0.0822	0.0753	-8.4
Other NCR Cities	0.0920	0.0764	-17.0
Rest of the Philippines	0.1261	0.0999	-20.8

Across all regions, birth rates declined over time. However, the decline was smallest in Manila (−8.4%)—the only jurisdiction directly affected by the contraceptive ban—compared to other NCR cities (−17.0%) and the rest of the Philippines (−20.8%). These descriptive trends suggest that although fertility fell nationwide, the reduction in Manila was markedly smaller. This relative divergence aligns with the regression-based findings, which pointed to a potential dampening of the fertility decline in Manila following the implementation of EO 003. The smaller post-2000 decline in birth rates supports the interpretation that the policy may have altered fertility trajectories in ways not observed elsewhere. If EO 003 influenced fertility behavior, we would expect birth rates in Manila to decline more slowly than in unaffected areas—and the data appear consistent with that expectation.

I recognize that any comparison involving the rest of the Philippines must account for the sheer size and heterogeneity of this group, which spans highly urbanized centers as well as rural and remote provinces. Aggregating such diverse regions may obscure meaningful local variation and exaggerate the contrast with Manila. That said, if Executive Order No. 003 had a substantive effect on fertility behavior, I would still expect fertility trends in Manila to diverge from those in the rest of the country—a pattern that both the DHS regressions and census-based birth rates consistently support.

In addition, several important caveats apply to the birth rate approximation. First, the method omits both child and maternal mortality, as well as any migration that may have occurred between the time of birth and the census year. Second, the computation restricts the sample to women who appear as household heads or as spouses of household heads, to increase the likelihood that observed children are their biological offspring. This restriction introduces composition effects. For instance, a 26-year-old woman listed as a household head in the census may differ systematically from a 25-year-old who assumed that role several years earlier. These differences are especially salient among younger women, where household formation often coincides with marriage and initial childbearing. As a

result, birth rates may show artificial dips in the census years themselves.

Despite these limitations, the estimates help build confidence in the plausibility of a fertility response to EO 003. They show that even when fertility is measured approximately from census structure, the direction of change aligns with expectations under a binding contraceptive ban. This, in turn, motivates the more rigorous analysis that follows, which uses DHS birth histories and econometric models to estimate fertility effects more precisely.

C. Reduced-Form Estimates: Effect of the Policy on Child Outcomes

Before proceeding to instrumental variables (IV) estimation, I begin by estimating reduced-form regressions to assess the intent-to-treat (ITT) effects of policy exposure on child outcomes. This step serves two purposes. First, it offers a preliminary test of whether the policy shock—treated as exogenous variation in contraceptive access—leads to measurable differences in child health. If no such differences emerge, any IV estimates may lack credibility or relevance. Second, the reduced-form regressions provide the denominator in two-stage least squares (2SLS), which estimates the total effect of the instrument without isolating its mechanism through an endogenous variable such as fertility.

The reduced-form specification follows a difference-in-differences framework. Formally, the reduced-form equation takes the following structure:

$$\text{Outcome}_{im} = \beta_0 + \beta_1 \text{Manila}_m + \beta_2 \text{ManilaPost}_{im} + \boldsymbol{X}'_{im}\gamma + \lambda_{y(i)} + \varepsilon_{im}$$

Where: Outcome_{im} is the child health outcome for child i of mother m (e.g., birth weight in grams or number of vaccine doses received); Manila_m is an indicator for residence in Manila; ManilaPost_{im} is an interaction term equal to 1 if the child was born in Manila in or after the year 2001 (i.e., the policy period), and 0 otherwise; $\boldsymbol{X}'_{im}$ includes individual- and household-level controls such as maternal age, maternal education, household wealth index, birth order, child sex, and twin status; $\lambda_{y(i)}$ denotes birth year fixed effects that flexibly capture national trends in child outcomes across cohorts; and ε_{im} is the error term, clustered at the primary sampling unit (PSU) level. The coefficient β_2 captures the reduced-form (intent-to-treat) effect of policy exposure on child outcomes.

Table **??** reports the results from reduced-form regressions estimating the intent-to-treat (ITT) effects of EO 003 exposure on child health outcomes. Each column presents a separate specification, with outcomes including birth weight, and number of vaccines received

The strongest and most consistent finding emerges in vaccination uptake. Exposure to the policy is associated with a significant reduction of 0.91 vaccine doses ($p < 0.001$). Given the Philippine Department of Health's recommendation of eight core immunizations by age one (Philippines Department of Health, 2023), this decline represents a shortfall of over 10% of the complete vaccination

Table 7—Reduced-Form Regressions: Intent-to-Treat Effects of EO 003 on Child Outcomes

	Birth Weight	Number of Vaccines
Manila × Post-2000	−349.597	−0.910***
	(0.142)	(<0.001)
Mother's Age	−35.581***	0.048***
	(<0.001)	(<0.001)
Mother's Education	−457.639***	0.047+
	(<0.001)	(0.061)
Wealth Index	−549.139***	−0.078**
	(<0.001)	(0.005)
Birth Order	182.550***	−0.241***
	(<0.001)	(<0.001)
Twin	−397.679*	0.117
	(0.016)	(0.489)
Child is Male	−7.062	−0.051
	(0.869)	(0.307)

+ p < 0.1, * p < 0.05, ** p < 0.01, *** p < 0.001
All regressions include birth year fixed effects.
Standard errors are clustered at the PSU level.
Estimates are weighted using DHS sample weights.

schedule. A child born in Manila in 2001—after the implementation of Executive Order No. 003—was, on average, likely to have missed nearly one routine vaccine compared to a counterpart born just a few years earlier or outside Manila. This gap could correspond to missing a third DPT booster, skipping the measles vaccine, or failing to complete the polio series—each of which increases exposure to otherwise preventable diseases.

In contrast, the estimated effect on birth weight is smaller and not statistically significant. On average, children born under the policy weighed 349.6 grams less than their peers in unaffected areas (p = 0.142). While the estimate does not reach statistical significance, its magnitude is nontrivial: a shortfall of nearly 350 grams could shift a child from normal birth weight into the low birth weight category (<2,500g), which has been linked to higher risks of infant morbidity and developmental delays (Katz et al., 2013).

Several covariates exhibit statistically significant associations with early-life health outcomes. Maternal age shows a negative association with birth weight (−35.6g per year, p < 0.001), which is consistent with clinical evidence linking advanced maternal age to elevated risks such as placental insufficiency and gestational complications (Lean, McCallum and Stevens, 2023). In contrast, each

additional year of maternal age corresponds to a modest increase in vaccine uptake (+0.048 doses, $p < 0.001$), which might suggest greater maternal experience or improved navigation of health systems (Barker, Yao and Mohammed, 2021). Meanwhile, maternal education shows a split pattern. Children of more educated mothers weigh less at birth (−457.6g, $p < 0.001$) but receive slightly more vaccines (+0.047 doses, $p = 0.061$). These results may reflect reporting differences: highly educated mothers may recall vaccination events more precisely and assess birth size more conservatively, which result in downward bias in reported weight (Filmer and Pritchett, 2001). Similarly, wealth index also exhibits statistically significant associations, though the direction diverges from conventional expectations. Higher wealth correlates with lower birth weight (−549.1g, $p < 0.001$) and fewer vaccine doses (−0.078, $p = 0.005$).

On the other hand, birth order has opposing effects. Later-born children weigh more (+182.6g, $p < 0.001$) but receive fewer vaccines (−0.241 doses, $p < 0.001$), which support theories of postnatal resource dilution within larger families (Black et al., 2008).Furthermore, twins weigh significantly less than singletons (−397.7g, $p = 0.016$), which is consistent with evidence of growth constraints in multiple gestations (Klebanoff, 2005). However, twin status shows no statistically significant effect on vaccination. Finally, child sex carries no meaningful association with either outcome. Male children weigh slightly less (−7.1g) and receive fewer vaccines (−0.051 doses), but both estimates are small and statistically insignificant ($p = 0.869$ and 0.307), which suggest negligible gender disparities in early-life health inputs in this context.

Taken together, these reduced-form estimates provide preliminary support for a quantity–quality trade-off in early-life investments. However, because exposure to the fertility shock (i.e., EO 003) is jointly determined with family size, these estimates conflate the effects of sibship size with the direct effects of policy exposure and local health service constraints. To isolate the causal impact of family size per se, I turn to an instrumental variable approach in the next section.

D. Second-Stage Estimates: The Quantity–Quality Trade-off

Having established preliminary intent-to-treat (ITT) effects of policy exposure on child outcomes through reduced-form regressions, I now turn to instrumental variables (IV) estimation to assess the causal effect of sibling exposure on early childhood health. The IV approach allows for identification of the total effect of having more siblings at birth on outcomes such as birth weight and immunization, to address the potential endogeneity of fertility decisions. Specifically, fertility may be jointly determined with unobserved factors such as household preferences, or income shocks, which would bias ordinary least squares (OLS) estimates.

To address this concern, I instrument the number of living siblings at birth using a policy-induced fertility shock: the interaction between residence in Manila and the post-2000 period (i.e., after the issuance of Executive Order No. 003). This interaction captures exogenous variation in fertility arising from localized

restrictions in contraceptive access. The identifying assumption is that, conditional on covariates and year fixed effects, this policy-driven variation affects child outcomes only through its effect on sibling exposure.

The IV model is estimated using two-stage least squares (2SLS), where the first stage regressed sibling exposure on the instrument and controls, and the second stage regresses child outcomes on the predicted sibling exposure. The estimating equation is of the following form:

$$\text{Outcome}_{im} = \beta_0 + \beta_1 \widehat{\text{Siblings}}_{im} + \boldsymbol{X}'_{im}\gamma + \lambda_{y(i)} + \varepsilon_{im}$$

Where Outcome_{im} is the child health outcome for child i of mother m; $\widehat{\text{Siblings}}_{im}$ is the instrumented number of living siblings at birth; $\boldsymbol{X}_{im}$ includes individual- and household-level controls (maternal age, maternal education, wealth, birth order, sex, twin status); and $\lambda_{y(i)}$ denotes birth year fixed effects. Estimates are weighted using DHS sample weights, and standard errors are clustered at the primary sampling unit (PSU) level.

Table~**??** presents instrumental variables (IV) estimates of the effect of sibling exposure—measured by the number of living siblings at birth—on two key early childhood outcomes: birth weight and vaccine uptake. These results represent an important step toward isolating the causal impact of fertility changes, building on the intent-to-treat estimates previously shown in Table 7.

Although none of the coefficients on sibship size reach statistical significance, the point estimates are large, negative, and consistent with the quantity–quality trade-off hypothesis. Specifically, an additional sibling is associated with an estimated 8,676-gram reduction in birth weight and a decrease of 23 vaccine doses. These magnitudes, while clearly inflated due to the wide standard errors and the weakness of the instrument, nevertheless suggest that if fertility expands in response to exogenous shocks, there may be meaningful consequences for child health. The lack of precision does not negate the possibility of real effects—it merely limits our ability to detect them definitively in this sample. Importantly, the sign and direction of the effects mirror those in the reduced-form estimates from Table 7, which strengthens the plausibility of the underlying mechanism: policy-driven increases in fertility may stretch household resources in ways that reduce investments per child.

The control variables provide additional insight into household and maternal characteristics that shape child outcomes. Birth order, for instance, is positively associated with both birth weight and vaccine uptake, which suggest that later-born children in this sample may benefit from parental learning or behavioral adaptation, even in the presence of additional siblings. In contrast, being a twin is associated with over 1,100 grams less in birth weight—a biologically expected result—while also linked to lower vaccination, which may reflect both logistical barriers and prioritization decisions in constrained households. The negative association between maternal age and birth weight could reflect accumulated health risks or socioeconomic selection among older mothers, while the small and impre-

Table 8—Instrumental Variables Regressions: Effect of Sibling Exposure on Child Outcomes

	Birth Weight	Number of Vaccines
Living Siblings at Birth	−8676.020	−22.968
	(0.324)	(0.265)
Mother's Age	−209.721	−0.412
	(0.236)	(0.319)
Mother's Education	−168.554	0.813
	(0.568)	(0.240)
Wealth Index	−754.195***	−0.625
	(<0.001)	(0.204)
Birth Order	1089.466	2.155
	(0.236)	(0.316)
Twin	−1169.464	−1.887
	(0.148)	(0.310)
Child is Male	140.637	0.352
	(0.407)	(0.386)

+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Instrument: Manila × Post-2000

All regressions include birth year fixed effects.

Standard errors are clustered at the PSU level.

Estimates are weighted using DHS sample weights.

cise effects of maternal education and wealth on vaccination uptake indicate that formal schooling and household resources alone do not guarantee equal access to preventive care.

One surprising result is the strongly negative and statistically significant association between the wealth index and birth weight (−754 grams, $p < 0.001$). This counterintuitive finding may reflect residual confounding or differences in reporting accuracy, but it also invites further investigation into whether urban wealth, where the cost of living is higher and stress exposure more intense, might offset the nutritional advantages typically associated with wealth in rural settings. Taken together, the IV estimates do not offer causal confirmation of the fertility–health linkage, but they suggest that the hypothesized trade-off remains empirically relevant. The patterns observed are coherent with theoretical expectations and underscore the importance of future work using stronger instruments or alternative policy shocks to credibly identify the full effect of family size on child outcomes.

VI. Limitations and Future Work

As with any empirical study, my analysis has important limitations. First, although I use an instrumental variable approach to address endogeneity in sibship size, the identification strategy rests on the assumption that policy exposure influences child outcomes only through its effect on fertility. While I believe this is a reasonable assumption given the policy context, I am unable to fully rule out other pathways—such as maternal stress or reduced postnatal care access—that may also be at play.

Second, my analysis is constrained by the limitations of the Demographic and Health Surveys (DHS). Key aspects of early childhood development—such as stunting and wasting—are not available in the DHS data I used, which restricts the scope of health outcomes I can examine. This gap points to a promising direction for future work: utilizing the Philippine National Nutrition Survey (NNS), which contains more detailed anthropometric indicators. Similarly, while I measure vaccination uptake by counting the number of doses received, I am unable to determine whether those vaccines were administered on schedule or if the child completed the full recommended series by age one. This distinction is important because delays in vaccination can reduce effectiveness and leave children vulnerable during critical developmental windows. Since some surveys do record vaccine dates, I hope to explore these timing dynamics more closely in future analyses.

Third, some of the outcomes I analyzed—especially perceived birth size—are based on maternal reports, which may be shaped by cultural expectations or subject to recall bias. Even birth weight, which is more objective, is missing for a share of children, especially in earlier survey waves or rural settings. This raises concerns about selection bias because the children with available data may differ systematically from those without. As I discussed earlier in the reduced-form results, there's a notable disconnect between reported birth size and measured birth weight. This discrepancy warrants closer attention—not just because it may signal reporting bias, but also because it might reflect how mothers perceive and evaluate infant health. Future work could investigate whether these gaps differ systematically across groups, such as by maternal education, geographic region, or socioeconomic status, and what that might reveal about prevailing health knowledge and beliefs.

Finally, the setting of Executive Order No. 003 in Manila limits how far these results can be extended to other contexts. The policy was geographically specific and politically shaped, with uneven implementation across barangays and varying access to reproductive services. Dumas and Lefranc (2019) noted that informal restrictions on contraceptive access in Manila began as early as 1997, well before the official issuance of Executive Order No. 003 in 2000. Even after its formal implementation, the policy's enforcement was uneven—varying significantly by barangay and often shaped by the discretion of local health officials. These inconsistencies further complicate efforts to isolate the policy's timing and intensity. These features make it difficult to apply the findings to places without

similar institutional and demographic conditions. Although I include survey-year fixed effects, the DHS data structure does not allow me to follow the same clusters over time, which restricts my ability to test for pre-policy trends or conduct spatially detailed robustness checks.

Future research can address these limitations by combining experimental or quasi-experimental variation in fertility with longitudinal administrative data on health. Replicating this framework in other natural experiments—such as decentralization-driven contraceptive rollouts or fertility shocks following natural disasters—would test the robustness and generalizability of the quantity–quality trade-off. In doing so, future work can help inform integrated policy approaches that explain how families adjust their investments when fertility changes.

VII. Conclusion

This study investigates the impact of exogenous fertility shocks on early childhood outcomes using the case of Executive Order No. 003 in Manila, which imposed restrictions on access to modern contraceptives beginning in 2000. Exploiting this localized policy change as a natural experiment, I employ both reduced-form regressions and instrumental variables (IV) estimation to assess whether increased sibling exposure—proxied by the number of living siblings at birth—affects child health investments and outcomes, specifically birth weight and vaccine uptake.

The reduced-form results show that exposure to the policy is significantly associated with lower vaccination coverage, and directionally negative—though imprecise—for birth weight. These findings suggest that the fertility increase induced by the policy may have had measurable consequences for children's health inputs. The IV estimates, while statistically insignificant due to weak instrument strength, are consistent in sign and magnitude with the reduced-form results. Point estimates suggest large negative effects of sibling exposure on both outcomes, and provide suggestive—though not definitive—evidence in support of the quantity–quality trade-off framework. The consistency across models reinforces the possibility that, under resource constraints, an exogenous rise in fertility may reduce parental investments per child.

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