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ABSTRACT

We study the intergenerational persistence of inequality by estimating grandmother-mother associations in the loss of a child, using pooled data from 119 Demographic and Health Surveys in 44 developing countries. Compared with compatriots of the same age, women with at least one sibling who died in childhood face 39% higher odds of having experienced at least one own-child death, or 7 percentage points at age 49. Place fixed effects reduce estimated mortality persistence by 47%; socioeconomic covariates explain far less. Within countries over time, persistence falls with aggregate child mortality, so that mortality decline disproportionately benefits high-mortality lineages.

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1 Introduction

Classic theoretical models of economic development study whether inequality persists across generations and how it interacts with the process of aggregate growth (Banerjee and Newman, 1993; Galor and Zeira, 1993). However, the scarcity of multigenerational data on income, consumption, and wealth in developing countries has hindered efforts to characterize intergenerational mobility.[1] Using multigenerational data from 44 developing countries, we estimate intergenerational persistence in a separate determinant of wellbeing—the death of a child—and study how it changes with aggregate progress against child mortality.

We rely on survey data to estimate associations between grandmothers and mothers in the loss of a child. Our analysis hinges on the coexistence, in a single survey, of a sibling history module—which asks a woman for survival information on all of her mother's children—and a birth history module—which asks for the same on her own children. In such a survey, one can estimate mortality persistence as the relationship between sibling and own-child mortality. To this end, we assemble the 119 Demographic and Health Surveys with both modules, providing data on 2.6 million births to 1.3 million women in contexts spanning varying levels of socioeconomic development and varying stages of the mortality transition.

The data reveal significant intergenerational persistence in the loss of a child. Within a country at a given age, women with at least one sibling who died under 5 face 39% higher odds of losing at least one child under 5. In absolute terms, the risk gap accumulates to 7 percentage points for women in their late 40s. At the child level, we find that the odds of dying under 5 rise 9% with each additional maternal sibling under-5 death, or 1 percentage point of risk. We refer to percentage changes in odds as *proportional* persistence and percentage point changes in risk as *absolute* persistence.

The interpretation of these large magnitudes depends on one's exact interest in mortality persistence. If one cares about the intergenerational persistence of life chances, then persistent

[1] Recent work focuses on educational mobility, using survey reports of own and parental education or census data on coresident parents and children (Torche, 2014; Neidhöfer et al., 2018; Narayan et al., 2018; Asher et al., 2020; Alesina et al., 2021; Muñoz, 2021).

mortality *risk* is more relevant than persistent mortality *outcomes*. The same is true if one views child mortality as a proxy for other forms of family disadvantage. The observed death of a sibling is an inherently noisy proxy for mortality risk or the socioeconomic determinants of mortality. This noise implies that our estimated outcome association understates the underlying risk association.[2] However, if one cares about the joint experience of sibling and maternal bereavement, then the outcome association is directly of interest.

Child mortality may persist across generations because of genetic inheritance, cultural transmission, socioeconomic immobility, the long reach of maternal childhood health, or geographic health disparities. To shed light on some of these channels, we conduct a simple accounting exercise that can be valuable for future work that seeks to identify causal mechanisms, analogous to how development accounting informs research into the mechanisms of economic development (Caselli, 2005). The data allow us to assess the last three channels by adding covariates and fixed effects to the mortality persistence regression. We find that place fixed effects account for 47% of mortality persistence, while covariates for maternal human capital, wealth, and maternal health account for far less. The fixed effects are based on place of residence, so in principle, they may reflect spatial sorting rather than place effects *per se*. However, we find that place fixed effects also halve mortality persistence in a subsample of women who have lived in the same place all their lives, casting doubt on the sorting explanation. More likely, the place fixed effects reflect the persistence of place—including disease ecology, public health infrastructure, and health care access—or perhaps unmeasured dimensions of human capital and wealth. Separate from these channels, we also find that neither mothers' nor grandmothers' fertility mediates persistence in cumulative child deaths experienced over the lifecycle.

Aggregate mortality decline may magnify or reduce mortality persistence, depending on its distribution across high- and low-mortality lineages. This link has close analogies in the Kuznets Curve (Kuznets, 1955), which describes how income inequality changes with income

[2]Vaupel (1988) makes the similar point that the intergenerational association of lifespan understates the intergenerational association of relative mortality hazards.

growth, and the Great Gatsby Curve (Krueger, 2012), which describes how intergenerational income mobility relates to income inequality. To assess it, we estimate mortality persistence separately by country and the child's five-year birth period. We then regress the estimates on UN under-5 mortality rates, with country and birth period fixed effects. We find that as aggregate mortality declines within a country over time, absolute mortality persistence falls, but proportional mortality persistence does not. Our results suggest that the decline of child mortality in the late 20^{th} and early 21^{st} centuries had greater absolute benefits for lineages with historically higher child mortality, but not enough to close their relative disadvantage.

Analysts of sibling history data have long worried that respondents omit deceased siblings (Helleringer et al., 2014). Based on independent responses from coresident mothers and daughters, we estimate that respondents underreport sibling childhood deaths by 11%. However, we find in Monte Carlo simulations that even if sample-wide underreporting were twice as extensive, bias in our estimators of mortality persistence would be small.

Our research contributes to a multidisciplinary literature on intergenerational associations in mortality and health. Biodemographers and behavioral geneticists have long studied family-level variation in longevity (Cohen, 1964; Vaupel, 1988; Herskind et al., 1996; Iachine et al., 2006), finding moderate heritability. Economists have until recently focused on early-life health, finding associations between mothers' height and children's health in poor countries (Venkataramani, 2011; Bhalotra and Rawlings, 2013), and between mothers' and children's birth weights' in rich countries (Black et al., 2007; Currie and Moretti, 2007; Royer, 2009).[3] More recent work in economics includes adult morbidity (Halliday et al., 2021) and longevity (Black et al., 2022) in rich countries. Scaled appropriately, our estimates of child mortality persistence are comparable to US intergenerational associations in birth weight and lifespan but smaller than that in adult morbidity.

The intergenerational persistence of child mortality also relates to two literatures on the welfare consequences and policy implications of multidimensional inequality. Recent work

[3]The birth weight studies find that early-life conditions rather than genetics account for much of the intergenerational correlation.

builds quantitative tools to capture the contribution of mortality to cross-country welfare inequality (Becker et al., 2005; Fleurbaey, 2009; Fleurbaey and Gaulier, 2009; Jones and Klenow, 2016) and the prevalence of deprivation (Baland et al., 2021). Our results endorse extending these tools to study persistent within-country mortality inequality. An older literature documents that mortality can fall without economic growth, using China, Cuba, Kerala, and Sri Lanka as examples (Caldwell, 1986; Sen, 1999). Their experiences suggest that the policy options for combating persistent inequality may go beyond conventional tools like progressive taxation and redistribution. Public health programs, for example, may reduce geographic mortality dispersion and thus reduce persistent inequality in life chances. Bhalotra and Rawlings (2013) find that maternal height becomes less related to infant mortality as immunization rates rise. Yet they, like us, find little role for long-run income growth.

2 Data

We draw on the Demographic and Health Surveys (DHS), which interview women of childbearing age (15-49). The surveys include a birth history module, which logs the respondent's live births and their survival. A subset also includes a sibling history module, which logs the respondent's reports of her mother's live births and their survival. We use the 119 surveys with both modules, leading to a 44-country sample. The countries are concentrated in sub-Saharan Africa but are also scattered across other developing regions (Appendix Table A.1).

The surveys have data on 1.7 million women, but our analysis necessitates two major sample restrictions. First, we omit respondents without siblings because they are uninformative about mortality persistence. Second, we omit respondents under 20 years old because their sibships may be incomplete, and they are unlikely to have given birth at least 5 years before the survey. We apply two minor restrictions, omitting respondents missing key covariates and those older than the standard DHS maximum age of 49. These restrictions leave 1.3 million women aged 20-49. When we analyze birth-level data, we exclude births missing under-5 mortality status or occurring more than 20 years before the survey, leaving 2.6 million births.

Descriptive statistics on the 1.3 million women appear in Table 1. On average, respondents are 32 years old and have 4.9 years of education. Roughly two-thirds live in rural areas. Respondents have fewer children than their mothers: children ever born averages 3.4, while siblings ever born averages 5.7. This discrepancy reflects both fertility decline and incomplete childbearing for younger respondents.

The sibling histories detect substantial mortality, with 32% of respondents reporting at least one sibling under-5 death. Surprisingly, however, the birth and sibling histories show similar own-child mortality rates for respondents and their mothers. Respondents averaged 0.43 dead before age 5, their mothers 0.61. Dividing by children or siblings ever born, both imply under-5 mortality risk of 11-13% per birth. Given the decline of infant and child mortality since the mid-20^{th} century, the similarity may suggest that respondents underreport deceased siblings. An alternative explanation is that our sample of adult respondents necessarily overweights families with more surviving children.[4] We discuss reporting errors and their consequences in Section 4.2.

Figure 1 shows basic patterns over the lifecycle. Older age groups exhibit higher risk of any child death and a larger gap by sibling mortality status, reflecting the accumulation of exposure with age and higher child mortality in earlier cohorts. At 20-24, 13% of women with deceased siblings have experienced child loss, compared with 9% among women without. By 45-49, these shares grow to 52% and 40%, respectively; the absolute gap more than doubles. These gaps partly reflect cross-country and cross-cohort variation, as women with deceased siblings tend to be from high-mortality countries and cohorts. The shaded area isolates the weighted average of *within-survey* gaps, accounting for 65-70% of the overall gap. Comparing women of the same age in the same survey removes variation between countries and cohorts.

The widening absolute gap suggests a proportional model. Appendix Figure A.2 rescales the vertical axis to log odds, revealing constant proportional gaps across across age groups, with 56-63% higher odds for women with at least one sibling death, or 36-41% within sur-

[4]If child mortality risk were independent of sibship size, this downward bias would exactly counterbalance an upward bias from omitting the respondent, who survived childhood (Trussell and Rodriguez, 1990). In our data, however, sibling under-5 mortality rises with sibship size (Appendix Figure A.1).

vey. These results suggest that a proportional model will best capture the accumulation of differential child mortality risk over the lifecycle.

3 Methods

We estimate proportional models to accommodate lifecycle variation. At the woman level, we specify a generalized linear model of the form:

$$\eta_{js} = \gamma_s + \sum_a \alpha_a age_{js}^a + \beta_1 dead_{js} + \beta_2 sibs_{js} \tag{1}$$

for woman j in survey s. The response η_{js} is a function of *child* deaths, while the covariate $dead_{ms}$ is a measure of *sibling* deaths. To shed light on the role of grandmothers' fertility, we report results with and without controlling for the number of siblings ever born, $sibs_{js}$. For an exclusive focus on within-survey inequality, we include a survey-specific intercept γ_s. Finally, because risk cumulates over the lifecycle, we control flexibly for age by including single-year age indicators. We restrict age effects to be the same for all surveys to reduce computational burden but show in the Appendix that our main results do not change when we allow them to vary by survey.[5]

For our headline result, we estimate a logit regression relating the occurrence of any child death to the occurrence of any sibling death at the woman level. In terms of equation (1), η_{js} is the log odds of the mother experiencing at least one child death, and $dead_{js}$ is an indicator for the grandmother experiencing at least one child death. Of the specifications we run, this one has the clearest interpretation. It also allows us to predict the sign of any bias from underreporting of sibling deaths, since non-differential underreporting will attenuate the coefficient on the binary version of $dead_{js}$.

To understand the variation driving our main results, we estimate several model variants. First, to flexibly accommodate the changing distribution of sibling deaths, we also report

[5]The flexible specification effectively controls for country-specific cohort variation, since age and cohort are collinear conditional on survey year.

estimations in which $dead_{js}$ is the count of sibling under-5 deaths. Second, for insight into the role of the respondent's fertility, we compare a Poisson regression of the count of child deaths at the woman level with a logit regression of mortality at the birth level. If women from high-mortality lineages accumulate more own-child deaths because they have more children, then the estimated parameters from the woman-level Poisson model will be larger than those from the birth-level logit model. The Poisson model is another version of equation (1), in which η_{js} is the logarithm of the expected number of child deaths experienced by the woman. However, the birth-level logit model requires a new specification:

$$\eta_{ijs} = \gamma_s + \beta_1 dead_{js} + \beta_2 sibs_{js} \tag{2}$$

where η_{ijs} now refers to the log odds of under-5 death for birth i to mother j in survey s. We omit the mother's current age because it is no longer directly related to cumulation of risk.

These functional forms treat mortality persistence as proportional, such that the odds or expected count of child deaths are proportional to the occurrence or count of sibling deaths. Our main estimands are odds ratios and incidence rate ratios. Because absolute persistence is of independent interest, we also compute average marginal effects. All analyses of individual-level data use sampling weights rescaled to reflect each survey's contribution to the sample and cluster standard errors at the survey cluster (village or city block) level.

4 Results

4.1 Pooled Estimates

Pooled estimates of child mortality persistence appear in Table 2. The top of each panel reports exponentiated coefficients: odds ratios for the logit models and incidence rate ratios for the Poisson models. Average marginal effects appear at the bottom.

Panel A uses an indicator for at least one sibling under 5 death as the main covariate.

Column (1) indicates that having at least one deceased sibling is associated with a 39% increase in the odds of having at least one deceased child. To capture the cumulation of risk over the lifecycle, we compute the average marginal effect at the last age in the sample, 49. Women with deceased siblings are 7 percentage points more likely to have experienced a child death by the end of reproductive age. Our proportional model fits the cumulation of differential mortality risk over a woman's reproductive years well. Appendix Figure A.3 reports similar results in pooled and age-specific estimations, with odds ratios stable at roughly 1.4 over the lifecycle, and average marginal effects roughly doubling from the early-20s to the late-40s. Stable odds ratios imply expanding absolute gaps.

The remainder of Table 2 compares alternative models of child mortality persistence, with results suggesting that neither the woman's fertility nor her mother's explains child mortality persistence. All six models—the woman-level logit, the woman-level Poisson, and the birth-level logit, with the sibling death indicator (Panel A) or count (Panel B)—find significant persistence. Within each model, the estimate changes little when we include siblings ever born as a covariate, suggesting that grandmothers' fertility plays little role in the results. A comparison of the woman-level Poisson estimates (columns [3]-[4]) to the birth-level logit estimates (columns [5]-[6]) further suggests that mothers' fertility plays little role. The incidence rate ratios from the woman-level models are similar to the odds ratios from the birth-level models, implying that risk to a woman cumulates in proportion to her number of children. If mortality persistence partly operated through a correlation of family mortality risk with fertility, then the woman-level estimates would exceed the birth-level estimates.

Of these remaining estimates, that in Panel B, column (6) is most intuitive: how an additional sibling death relates to the odds of death for a given child, holding fixed the number of siblings. This specification is appealing because it holds family size fixed in both generations. It finds that each additional under-5 death of a mother's sibling is associated with a 9% increase in the odds of an own-child's death, or a 1 percentage point increase in the probability of death.

4.2 Interpretation and Robustness

Our results raise three concerns: about the magnitudes of the estimates, the conflation of intra- with inter-cohort variation, and bias from underreporting of sibling deaths.

Magnitudes The results in Table 2 are straightforward to interpret by themselves. But how does sibling death compare to other determinants of child mortality, and how does child mortality persistence compare with other intergenerational associations in health?

In our data, socioeconomic differentials in any child death somewhat exceed cumulative mortality persistence. Appendix Figure A.4 reports that the odds ratio for rural residence is 2.05, and that for not finishing primary school is 2.65, compared to the mortality persistence odds ratio of 1.39. To find a mortality persistence odds ratio above 2, one has to look to women from extremely high mortality families. Relative to women with no deceased siblings, women with eight or more have 2.29 the odds of losing at least one child. However, such women comprise less than 0.2% of the sample.

When we compare child mortality persistence to other intergenerational health associations in the US, we find that it is similar to the birth weight and lifespan associations but smaller than the adult morbidity association. Currie and Moretti (2007) find that children with low birth weight mothers are 3.9 percentage points more likely be low birth weight themselves, implying an odds ratio of 1.9. We find a larger marginal effect but smaller odds ratio. To obtain quantities that we can compare to intergenerational associations in lifespan and adult morbidity, we compute partial correlations of sibling and own-child death, conditional on survey indicators (Appendix Table A.2).[6] The correlation in any death is 0.08, and the correlation in the count of deaths is 0.07. These magnitudes are similar to Black et al.'s (2022) estimate of 0.09 for the mother-daughter correlation in age at death and somewhat smaller than Halliday et al.'s (2021) estimates of intergenerational associations in adult morbidity (proxied by self-reports of health status and health conditions), which imply mother-daughter correlations of 0.11-0.17.

[6]To avoid issues of proportionality over the lifecycle, we limit this analysis to women aged 45-49.

Cohort Effects Our estimations include survey indicators but not cohort indicators, so they mix within-cohort comparisons of women from low- and high-mortality families with between-cohort comparisons of women from low- and high-mortality cohorts. Our woman-level models include age indicators, but because the data were collected in a variety of times and places, the age indicators do not fully absorb sample-wide cohort variation, let alone country-specific cohort variation. Appendix Figure A.5 adds interactions of survey indicators and age group indicators to all 12 models in Table 2, finding no change in the estimates.[7] The survey-by-age group effects effectively control for country-specific cohort variation, so our results do not reflect comparisons of cohorts with high and low sibling mortality. Instead, they are primarily driven by within-cohort comparisons.

Reporting Errors Respondents may underreport the childhood deaths of their siblings. To assess the extent of underreporting, we follow Masquelier and Dutreuilh (2014) in studying coresident mothers and daughters. The DHS instructs enumerators to interview each 15-49 year old female household member alone, so a mother's birth history provides an independent check on her daughter's sibling history. Linkage of mother and daughter respondents is possible in 85 of the 119 surveys; in these surveys, 38% of 15-19 year olds coreside with their 30-49 year old mothers. In these mother-daughter pairs, daughters' reports of sibling under-5 deaths are highly correlated with mothers' reports of child under-5 deaths ($\rho = 0.90$), with moderate underreporting and minimal overreporting (Appendix Tables A.3-A.4). When mothers report no child deaths, 98% of daughters report no sibling deaths. When mothers report at least one child death, 86% of daughters report at least one sibling death, with the exact counts matching in 77-80% of cases. Overall, daughters report 11% fewer deaths than mothers.

Bias from underreporting depends on whether we use the count of sibling deaths or an indicator for any sibling death. For the indicator, if underreporting is nondifferential, then it biases us toward finding no persistence (Davidov et al., 2003; Mahajan, 2006).[8] For the

[7] We use five-year rather than one-year age groups in these interactions because the latter proved computationally burdensome.

[8] However, women who erroneously report having no siblings drop out of the sample, which may have

count, the bias is more difficult to characterize analytically (Bound et al., 2001). Due to these ambiguities, we perform Monte Carlo simulations of a plausible form of measurement error. We simulate the omission of deceased siblings assuming a fixed probability of omission per deceased sibling. After each draw, we drop observations with 0 reported siblings and estimate the 12 regressions in Table 2. We assess how the estimates change as we increase the probability of omission from 0 to 25% (over twice the rate in the mother-daughter pairs).

Even at large probabilities of omission, the simulated biases in the mortality persistence estimates are small (Appendix Figure A.6). For the mother-level regression of any child death on any sibling death, a 25% probability of omission leads to a mean odds ratio of 1.378, compared to our original result of 1.386. For the birth-level regression of child death on the number of sibling deaths and siblings ever born, a 25% probability of omission leads to a mean odds ratio of 1.105, compared to our original result of 1.092. The simulations suggest that our estimations are robust to underreporting.

4.3 Accounting for Mortality Persistence

What channels account for intergenerational persistence in child mortality, and how does it vary within and between populations? We assess channels by adding covariates, and we explore heterogeneity by estimating persistence separately by country and gender. The analysis of gender heterogeneity requires birth-level data, so we use the birth-level logit specification from Table 2, Panel B, column (6) throughout.

Adding covariates Potential explanations for intergenerational persistence in child mortality include socioeconomic immobility, geographic health inequality, cultural transmission, health transmission, and genetic inheritance. To shed light on some of these explanations, Table 3 adds covariates and fixed effects to the regression. Our analysis is limited by the scope of the DHS questionnaire, which collects no genetic information and asks only a handful of questions on socioeconomic, geographic, and adult health outcomes. We account for place

different consequences from misclassification alone.

by including survey cluster (village or city block) fixed effects in a conditional logit model. Because the conditional logit model requires outcome variation within all units, we omit clusters lacking variation in child mortality from the analysis. For socioeconomic status, we use the mother's years of education and the DHS household wealth index, formed by taking the first principal component of a vector of indicators for durable goods ownership and improved housing conditions at the time of the interview. For maternal health, we use the mother's height. This accounting exercise is useful for pinpointing margins of interest for future research but not for directly identifying causal mechanisms.

Panel A finds that geography accounts for much of the intergenerational relationship, while measured socioeconomic status accounts for fall less. Column (1), which adds no further covariates or fixed effects, finds an odds ratio of 1.085, similar to Table 2, Panel B, column (6). Column (2) adds survey cluster fixed effects, shrinking the odds ratio to 1.043, a 49% reduction toward unity. Further adding the mother's education and the household's wealth index (column [3]) results in an odds ratio of 1.039, only 14% smaller. Thus, women with siblings who died in childhood live in places with higher under-5 mortality rates, which explains nearly half of the increased mortality odds their own children face. Place may capture disease ecology, public health infrastructure, and health care access, but also omitted socioeconomic variables. However, measured socioeconomic variables account for a limited share of the remaining persistence of child mortality.

Place contributes to health inequality (Burstein et al., 2019) and socioeconomic immobility (Asher et al., 2020; Alesina et al., 2021; Muñoz, 2021), so its primacy in the intergenerational persistence of child mortality is not surprising. Nevertheless, because the fixed-effect models condition on *current* place, whether unhealthy places are inherited in childhood or attained in adulthood through residential sorting is unclear. The DHS has limited information on childhood place of residence, but 77 of the 119 surveys in the sample ask whether the respondent has lived in her current place of residence all her life. To partially assess the role of residential sorting, we compare estimates with and without cluster fixed effects using data on only non-

migrants, who constitute 44% of the surveys with information on migrant status. The sample restriction does not entirely solve the interpretation issue, since the non-migrant subsample is self-selected, but similar results in the non-migrant subsample and the full sample would suggest a role for the inheritance of place in childhood.

Panel B analyzes non-migrants from the 77 surveys with information on migrant status, finding little change from the full sample results. Among non-migrants living in survey clusters with variation in child death, the addition of cluster fixed effects shrinks the odds ratio from 1.073 to 1.041, so that place accounts for 44% of mortality persistence. Given the resemblance to the full-sample result in Panel A, these findings suggest that the inheritance of unhealthy places accounts for half of the intergenerational persistence of child mortality.

To consider the role of maternal health, Panel C controls for the respondent's height in the 103 surveys with relevant data, finding that it explains little of the mortality association. Adult height is a proxy for early-life health health (Currie and Vogl, 2013) and the basis for Bhalotra and Rawlings' (2013) research on intergenerational health transmission. Although height negatively predicts child survival, it does not change the odds ratio on deceased siblings.[9]

Heterogeneity Appendix Figure A.7 reports birth-level odds ratios by country. Estimates are uniformly greater than 1, but with considerable heterogeneity, ranging from 1.02 to 1.24. Appendix Table A.5 investigates gender heterogeneity in the pooled birth-level model, estimating separate regressions for boys and girls, as well as splitting the count of sibling deaths into brother and sister deaths. We find little heterogeneity, with odds ratios of 1.08-1.11 for brothers, sisters, boys, and girls.

4.4 Mortality Persistence over the Mortality Transition

The country-level heterogeneity in Appendix Figure A.7 raises the question of how the intergenerational persistence of child mortality varies with aggregate health conditions. The answer

[9]Consistent with Bhalotra and Rawlings (2013), the conditional logit finds that a 10 centimeter increase in height predicts 10% lower odds, or a 1.4 percentage point lower probability, of under-5 death.

sheds light on the progressivity of mortality decline: whether previously high- or low-mortality lineages benefit more from aggregate health improvement. To investigate, we relate mortality persistence to the aggregate under-5 mortality rate. We consider mortality conditions at the time of each *child's* birth, as data on conditions at the time of the *mother's* birth are not uniformly available and are more subject to recall error.

The results of this exercise may depend on whether we measure mortality persistence in proportional or absolute terms. The relationship between between the odds ratio and the marginal effect changes as mortality falls in aggregate. Appendix Figure A.8 demonstrates this point by drawing the relationship between the marginal effect and the odds ratio for a binary covariate.[10] Holding baseline mortality fixed, the marginal effect increases with the odds ratio; holding the odds ratio fixed, the marginal effect increases with baseline mortality. At a higher mortality rate, a given proportional association implies a larger absolute association.

This three-way linkage of the aggregate mortality rate, the odds ratio, and the marginal effect implies that proportional mortality persistence and absolute mortality persistence need not move in the same direction during aggregate mortality decline. Both could decrease, both could increase, or the marginal effect could decrease while the odds ratio increases. The only impossibility is for mortality decline to be accompanied by a (weakly) rising marginal effect and a (weakly) falling odds ratio.

We assess which of these scenarios best characterizes child mortality decline in the developing world in the late 20^{th} and early 21^{st} centuries. Because we are interested in mortality decline, a within-country phenomenon, we construct a country-period panel of persistence estimates and merge it with UN country-period estimates of child mortality rates. To form this panel, we run the birth-level logit regression from Table 2, Panel B, column (6)—of child death on the number of deceased and ever-born siblings, conditional on survey indicators—for each country by five-year birth period cell.[11] The five-year periods correspond to when the

[10] If the covariate is multivalued, the relationship depends on its distribution.

[11] The birth-level data allow us to ask whether persistence parameters vary by year, and the count of sibling deaths captures the changing distribution of sibling mortality as mortality falls.

respondent gave birth, not when she was born.[12] Just as in the earlier birth-level analyses, each respondent may enter the sample multiple times. The analysis seeks to describe how the gains from mortality decline are distributed across women giving birth in a particular period.

We then estimate linear regressions of these estimated parameters on the contemporaneous under-5 mortality rate (from the UN), country fixed effects, and period fixed effects. In some specifications, we additionally control for the log of real annualized GDP per capita (from the Penn World Table) and an indicator for armed conflict (from UCDP/PRIO). The coefficient on the under-5 mortality rate represents how mortality persistence changes with overall mortality within a country over time. We cluster standard errors by country to account for within-country serial correlation.

Country-level time series of the under-5 mortality rate highlight two points (Appendix Figure A.9). First, the Rwandan genocide was a singular mortality event, with an under-5 mortality rate over 40% higher than the next highest. Because we are interested in secular decline rather than shocks, and because the extent of mortality during the genocide is extremely uncertain, we omit this episode from our sample.[13] Second, under-5 mortality predominantly trended downward in sample countries over the sample period. This result motivates our interest in the distribution of mortality decline.

Table 4 reports the two-way fixed effect estimates, finding that mortality decline is strongly associated with falling absolute persistence but only weakly associated with falling proportional persistence. A reduction in under-5 mortality of 0.1 (100 per 1000 live births) is associated with a weakening of the average marginal effect by 0.007, large relative to the mean average marginal effect of 0.009. If we run a regression of UN under-5 mortality on period and country fixed effects in our sample, the period fixed effects indicate that mortality fell on average by 0.152 (i.e., 152 per 1000 live births) over the 40 years in our sample. Multiplied by our coefficients, that change implies a decline in the average marginal effect by 0.010. In contrast,

[12]UN mortality data are available for all periods in which respondents gave birth but not for all periods in which respondents were born.

[13]Our series from the UN Population Division peaks at 466 per 1000 live births in 1990-4, while the UN Inter-Agency Group for Child Mortality Estimation series peaks at 276 in 1994 (`http://childmortality.org`).

log GDP per capita and armed conflict exhibit no relationship with mortality persistence.

At the same time, net of country and period fixed effects, the odds ratio is not significantly related to the under-5 mortality rate, although the coefficient is also positive. Aggregate mortality decline tends to benefit high-mortality lineages more than low-mortality lineages in terms of absolute bereavement risk. But in proportional terms, high-mortality lineages do not gain significantly on low-mortality lineages. In this sense, relative inequality in bereavement risk does not diminish during the process of aggregate mortality decline.

The Appendix extends Table 4 in two dimensions. First, Figure A.10 reports estimates of a semi-parametric version of the two-way fixed effect regression, in which the under-5 mortality rate enters as a series of bin indicators rather than a single linear term. The results show no substantial departures from linearity. Second, Figure A.11 re-estimates the regression leaving out one country at a time, showing that our estimates are not driven by any single country.

5 Conclusions

In data from a broad swath of the developing world, risk of losing a child is intergenerationally persistent. The fact of this persistence may not be surprising, but its magnitude is new, and it is large. Within a population at any given age, women with at least one sibling who died in childhood face 39% higher odds of losing at least one child. At the end of the childbearing period, these elevated odds translate to a 7 percentage point risk increase. This pattern appears to only partly reflect persistent inequality in socioeconomic variables. Geographic inequality plays a larger role in the perpetuation of child mortality risk across generations.

In studying child loss across generations, we contribute to a new demographic literature that shifts attention from deceased individuals to their survivors. Bereaved individuals face physical and mental health risks (Stroebe et al., 2007), and unequal distributions of mortality imply unequal distributions of bereavement. The cumulative toll of maternal bereavement is large; in some African countries, a majority of middle-aged women have experienced the death of at least one child under 5 (Smith-Greenaway and Trinitapoli, 2020; Alburez-Gutierrez

et al., 2021; Smith-Greenaway et al., 2021). The unequal burden of bereavement may be an important source of inequality in wellbeing, especially when it involves parental loss of a child (Li et al., 2003, 2005; Rogers et al., 2008).

The excess risk of child death among women who were bereaved of their siblings dissipates as child mortality falls in aggregate, suggesting a new way to think about the distribution of mortality decline. In absolute terms, high-mortality lineages benefited more from mortality decline. This result may be specific to the unprecedented, broad-based improvements in child health that many developing countries experienced in the late 20^{th} and early 21^{st} centuries. As the drivers of mortality decline shift from public health programs to individualized medicine, mortality decline may become less progressive.

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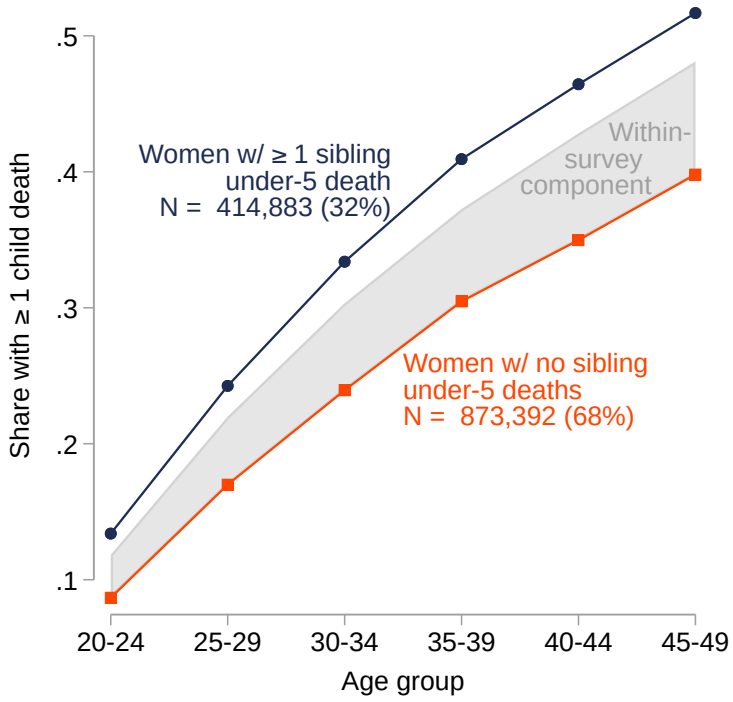
Table 1: Descriptive Statistics, Women Aged 20-49

	Mean	Std. Dev.
Age	32.00	8.28
Years of education	4.88	4.73
Rural residence	0.63	0.48
Siblings ever born	5.67	2.64
Siblings deceased under 5	0.61	1.15
At least one sibling deceased under 5	0.32	0.47
Children ever born	3.36	2.66
Children deceased under 5	0.43	0.90
At least one child deceased under 5	0.26	0.44
Observations	1,288,072	

Note: Sample includes women with at least one sibling ever born from 119 Demographic and Health Surveys in 44 countries. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Figure 1: Share with Any Child Death, by Any Sibling Death



Note: For each five-year age group, we plot the share of women with at least one child death separately for women with and without deceased siblings. The within-survey component is the weighted average of the difference in shares within in each survey. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Table 2: Pooled Estimates of Mortality Persistence

	Logit (Mother) Any child death		Poisson (Mother) # child deaths		Logit (Birth) Child death	
	(1)	(2)	(3)	(4)	(5)	(6)
A. Indicator of sibling death						
Any sibling U5 death	1.386	1.370	1.237	1.242	1.202	1.233
	[.0085]	[.009]	[.0058]	[.0062]	[.0069]	[.0077]
Sibs ever born		1.005		.998		.988
		[.0012]		[.00094]		[.0012]
AME(any sib. death)	.073	.070	.219	.224	.021	.024
Observations	1,288,072	1,288,072	1,288,072	1,288,072	2,609,862	2,609,862
B. Count of sibling deaths						
# sibling U5 deaths	1.141	1.141	1.085	1.093	1.075	1.092
	[.0027]	[.003]	[.0017]	[.002]	[.0022]	[.0025]
Sibs ever born		1.000		.992		.982
		[.0012]		[.00098]		[.0012]
AME(# sib. deaths)	.029	.029	.084	.091	.008	.010
Observations	1,288,072	1,288,072	1,288,072	1,288,072	2,609,862	2,609,862

Note: The reported estimates are logit odds ratios and Poisson incidence rate ratios. Brackets contain standard errors clustered at the survey cluster level. AME refers to the average marginal effect of sibling death(s); in the woman-level models, it is computed at age 49. All models include survey indicators. Woman-level models also include indicators for the woman's age in single years. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Table 3: Adding Covariates

	Logit with survey effects	Conditional logit with cluster effects	
	(1)	(2)	(3)
A. All, N = 2,397,677			
Sibs deceased under 5	1.085	1.043	1.037
	[.0025]	[.0024]	[.0024]
Sibs ever born	Yes	Yes	Yes
SES variables	No	No	Yes
B. Non-migrants, N = 491,640			
Sibs deceased under 5	1.073	1.041	1.038
	[.0047]	[.0051]	[.0051]
Sibs ever born	Yes	Yes	Yes
SES variables	No	No	Yes
C. Not missing height, N = 1,191,027			
Sibs deceased under 5	1.077	1.038	1.037
	[.0033]	[.0033]	[.0033]
Sibs ever born	Yes	Yes	Yes
Height	No	No	Yes

Note: This table reports odds ratios from birth-level logit regressions of under-5 child death on the number of under-5 sibling deaths, the number of siblings ever born, and the indicated explanatory variables. We omit clusters lacking variation in child mortality. Migrant status is available in 77 surveys; height is available in 103. Socioeconomic variables include maternal education and a wealth index based on principal component analysis over a vector of durable goods ownership indicators. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Table 4: Panel Analyses of Mortality Persistence over the Mortality Transition

	Average marginal effect				Odds ratio			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Under-5 mortality [0-1] (UN)	0.065			0.068	0.54			0.59
	[0.024]			[0.025]	[0.36]			[0.38]
Log GDPpc (PWT)		0.00044		0.0013		0.023		0.030
		[0.0016]		[0.0014]		[0.018]		[0.018]
Conflict [0/1] (UCDP/PRIO)			-0.0000072	0.00050			-0.0036	0.0019
			[0.0014]	[0.0013]			[0.014]	[0.012]
Mean of estimated parameter	0.009	0.009	0.009	0.009	1.092	1.092	1.092	1.092
Std. dev. of estimated parameter	0.008	0.008	0.008	0.008	0.090	0.090	0.090	0.090
Country-period cells	270	270	270	270	270	270	270	270

Note: Each observation is a country-period cell. The dependent variable is the cell-specific mortality persistence odds ratio (OR) or average marginal effect (AME) estimated from a birth-level logit regression of under-5 death on the mother's number of under-5 sibling death, the mother's number of siblings ever born, and survey indicators. All cell-level regressions include country and period fixed effects. Brackets contain standard errors clustered at the country level.

Table A.1: Demographic and Health Surveys in the Sample

Afghanistan: 2010, 2015	Lesotho: 2004, 2009, 2014
Bangladesh: 2001	Madagascar: 1992, 1997, 2004, 2009
Benin: 1996, 2008	Malawi: 1992, 2000, 2004, 2010, 2015
Bolivia: 1994, 2003, 2008	Mali: 1996, 2001, 2006, 2012
Burkina Faso: 1999, 2010	Morocco: 1992, 2003
Burundi: 2010, 2016	Mozambique: 1997, 2003, 2011
Cambodia: 2000, 2005, 2010, 2014	Namibia: 1992, 2000, 2013
Cameroon: 1998, 2004, 2011	Nepal: 1996, 2006, 2016
Central African Republic: 1994	Niger: 1992, 2006, 2012
Chad: 1997, 2004, 2015	Nigeria: 2008, 2013
Congo, Democratic Republic: 2007, 2013	Peru: 1991, 1996, 2000
Congo, Republic: 2005, 2011	Philippines: 1993, 1998
Côte d'Ivoire: 1994, 2012	Rwanda: 2000, 2005, 2010, 2015
Dominican Republic: 2002, 2007	São Tomé & Príncipe: 2008
Ethiopia: 2000, 2005, 2011, 2016	Senegal: 1993, 2005, 2011
Gabon: 2000, 2012	Sierra Leone: 2008, 2013
Ghana: 2007	South Africa: 1998, 2016
Guinea: 1999, 2005, 2012	Swaziland: 2006
Haiti: 2000, 2006, 2017	Tanzania: 1996, 2004, 2010, 2015
Indonesia: 1994, 1997, 2002, 2007, 2012	Togo: 1998, 2014
Jordan: 1997	Zambia: 1996, 2002, 2007, 2013
Kenya: 1998, 2003, 2009, 2014	Zimbabwe: 1994, 1999, 2005, 2010, 2015

Table A.2: Partial Correlations of Sibling and Child Under-5 Mortality, Women Aged 45-49

	Any child death and any sibling death	# child deaths and # sibling deaths
	(1)	(2)
Within-survey correlation	0.077	0.074
Number of observations	131,518	131,518

Note: Partial correlations are computed after conditioning on survey indicators. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Table A.3: Mothers' vs. Daughters' Reports of Any Under-5 Death

	D's report (%)		
M's report	0	1+	N
0	98.0	2.0	59,339
1+	14.4	85.6	35,552

Note: Sample includes coresident 15-19 year olds and their 30-49 year old mothers when both responded to the survey. Mothers and daughters are interviewed separately and privately. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Table A.4: Mothers' vs. Daughters' Reports of Any Under-5 Death

	D's report (%)							
M's report	0	1	2	3	4	5	6+	N
0	98.0	1.6	0.3	0.1	0.0	0.0	0.0	59,339
1	17.3	80.4	1.9	0.2	0.1	0.0	0.0	21,114
2	11.2	6.5	79.9	2.1	0.3	0.0	0.0	9,172
3	8.3	3.1	7.8	77.7	2.8	0.4	0.0	3,761
4	7.7	2.6	3.1	7.6	77.0	1.5	0.4	1,505

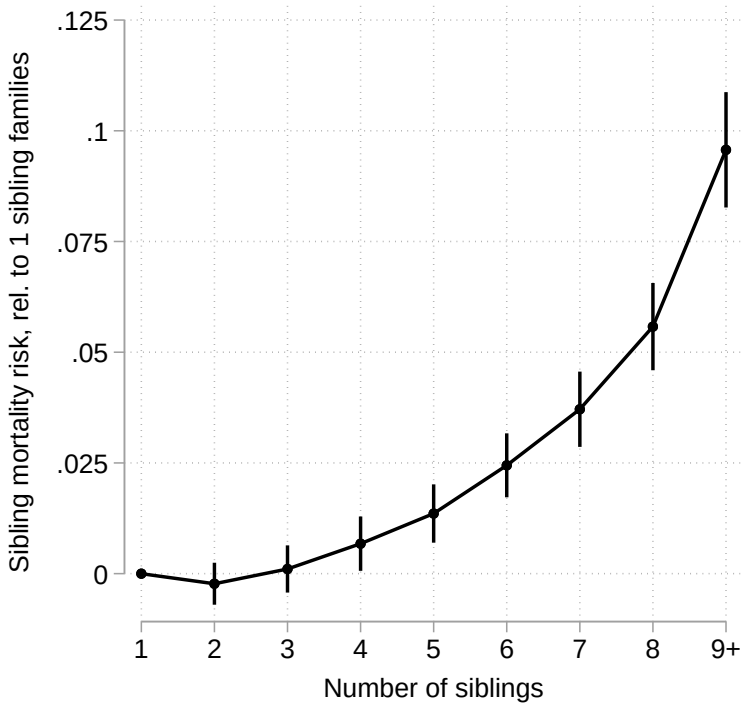
Note: Sample includes coresident 15-19 year olds and their 30-49 year old mothers when both responded to the survey. Mothers and daughters are interviewed separately and privately. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Table A.5: Pooled Birth-Level Logit Estimations by Gender

	Female	Male	Female	Male
	(1)	(2)	(3)	(4)
# sibling U5 deaths	1.09	1.09		
	[.0034]	[.0032]		
Sibs ever born	.98	.98		
	[.0016]	[.0015]		
# female sibling U5 death			1.11	1.09
			[.0058]	[.0056]
Female sibs ever born			.99	.98
			[.0022]	[.0021]
# male sibling U5 death			1.08	1.10
			[.0051]	[.0048]
Male sibs ever born			.98	.98
			[.0022]	[.0021]
AME(# sib. deaths)	.010	.010		
AME(# female sib. deaths)			.011	.010
AME(# male sib. deaths)			.009	.011
Observations	1,276,858	1,333,004	1,276,858	1,333,004

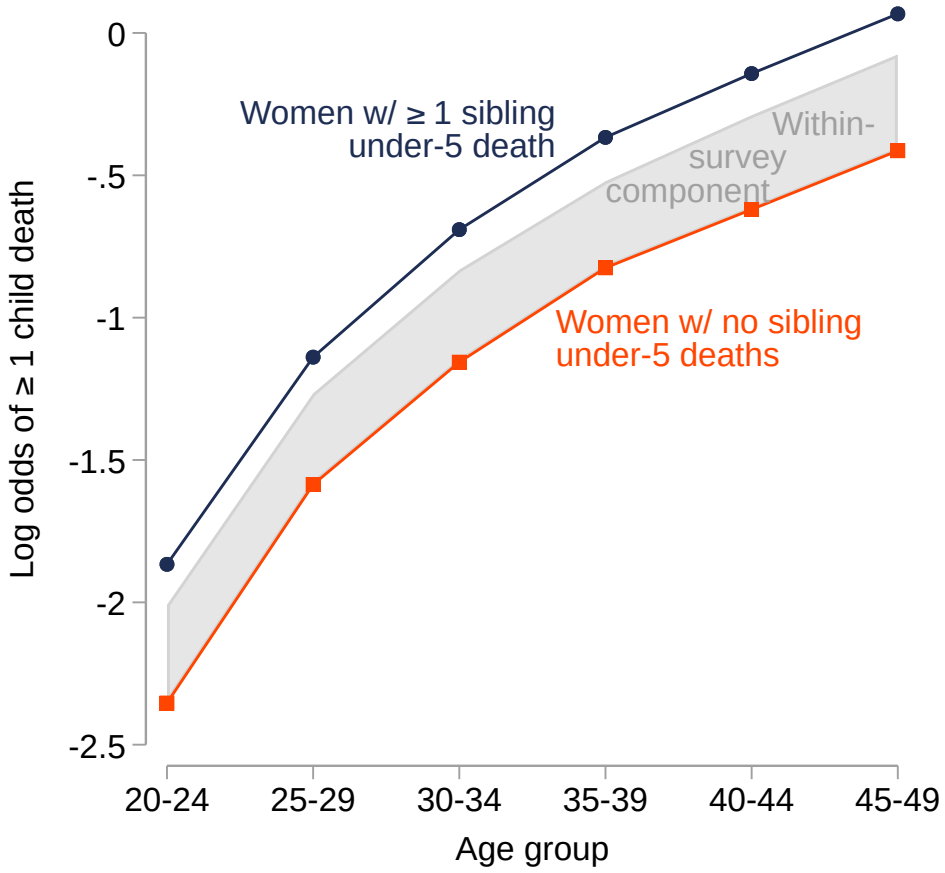
Note: The reported estimates are logit odds ratios. Brackets contain standard errors clustered at the survey cluster level. AME refers to the average marginal effect of the indicated measure of sibling death(s). All models include survey indicators. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Figure A.1: Sibship Size and Sibling Mortality



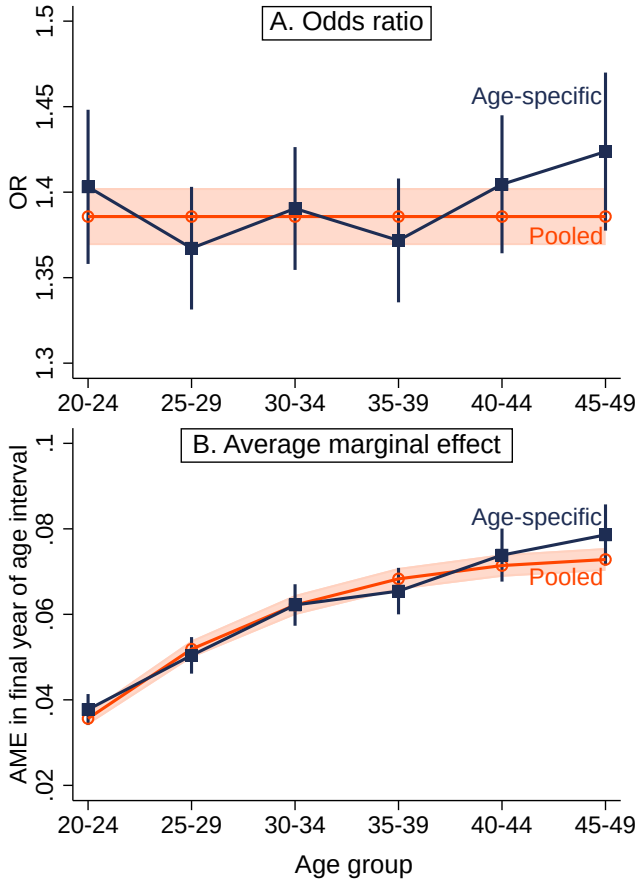
Note: This figure plots the relationship between sibship size and sibling mortality rates. The unit of observation is the survey-sibsize cell. We regress the sibling under-5 mortality rate on sibship size indicators and survey indicators. Mortality rates are scaled from 0 to 1. Cells are weighted by the number of women. Spikes are 95% confidence intervals based on standard errors clustered at the country level. Sampling weights are rescaled to reflect each survey's contribution to the cell.

Figure A.2: Log Odds of Any Child Death, by Any Sibling Death



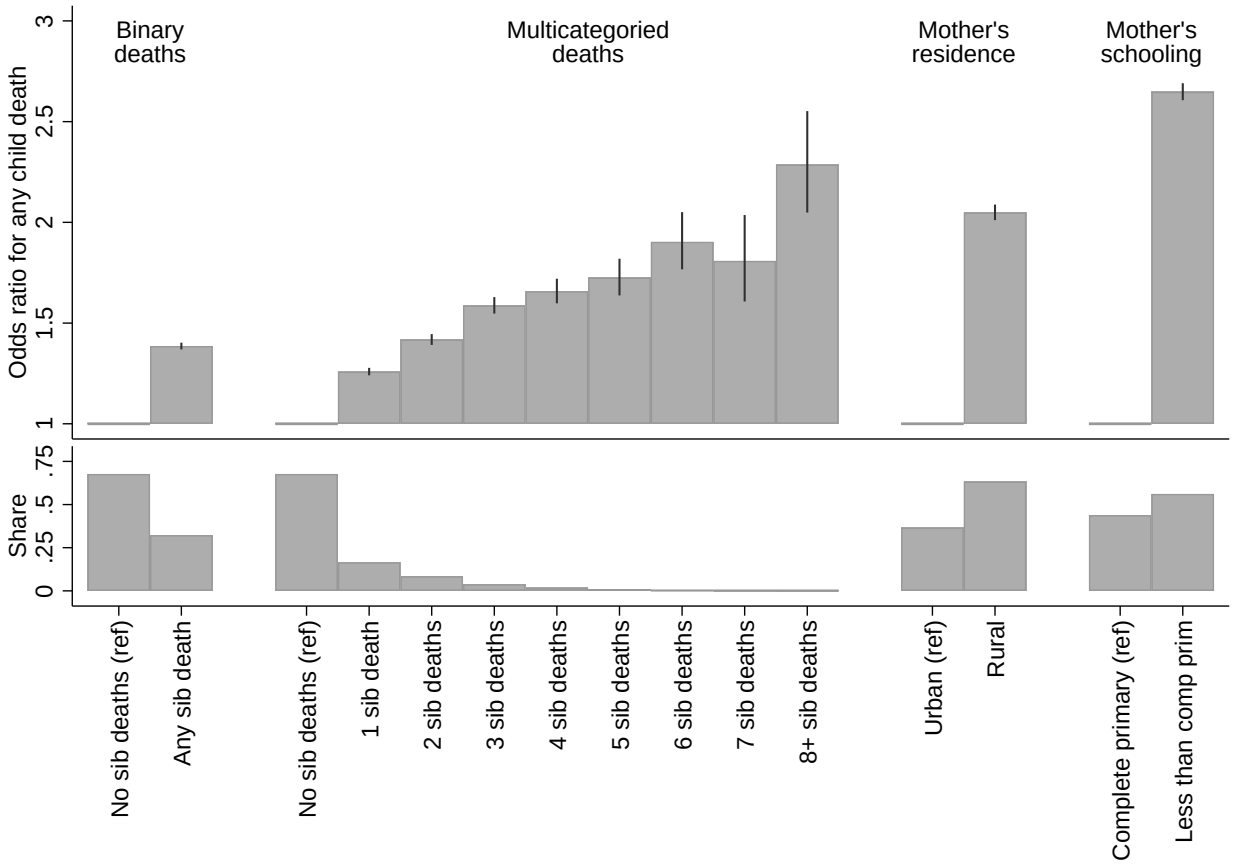
Note: For each five-year age group, we plot the log odds of any under-5 child death separately for women with and without deceased siblings. The within-survey component is calculated in log odds using the within-survey component and share of women with no sibling deaths in Figure 1. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Figure A.3: Mother-Level Logit Results by Age



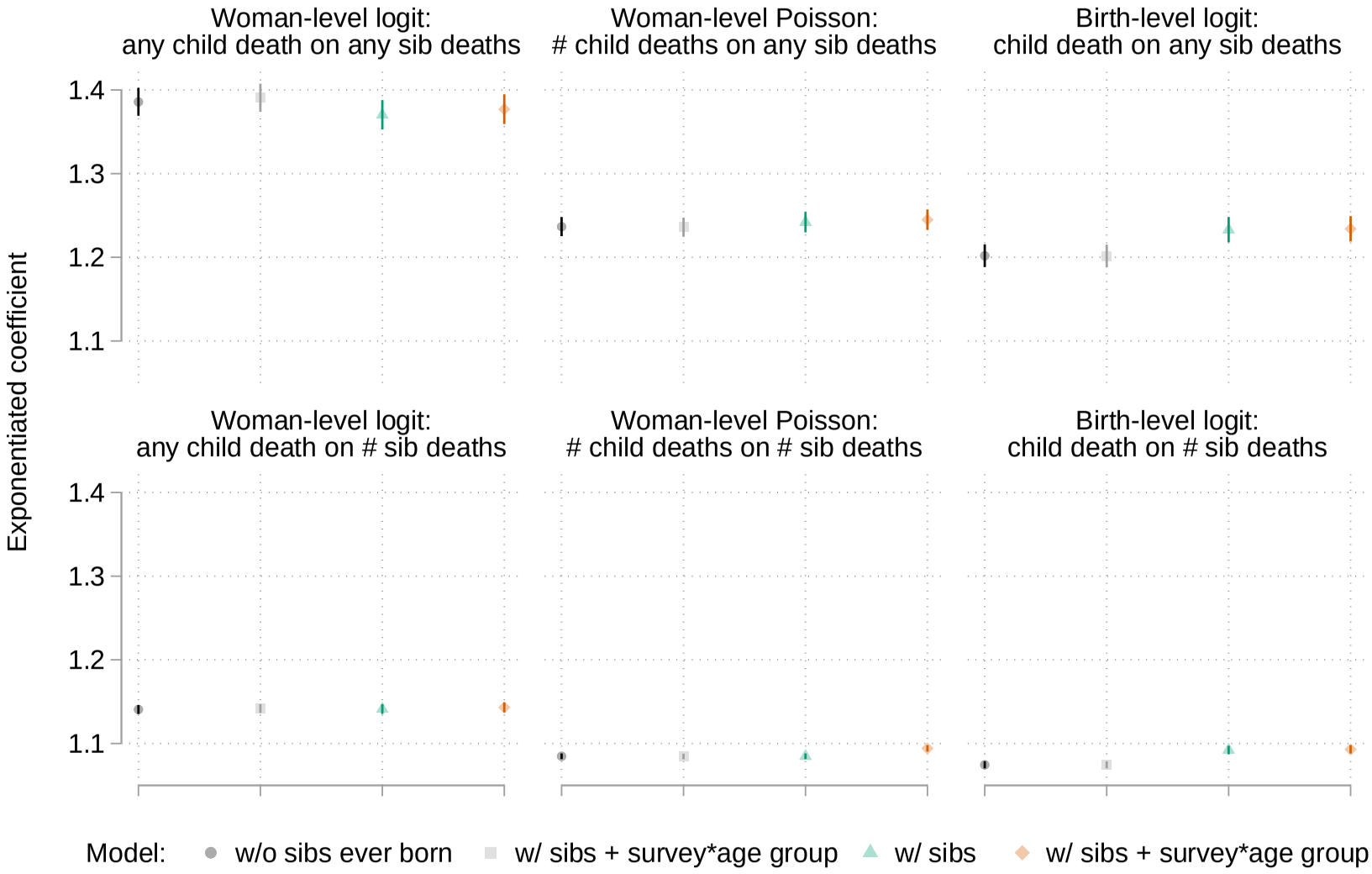
Note: This figure demonstrates the robustness of the woman-level logit estimates to age specific estimations. Point estimates and 95% confidence intervals based on women-level logit regressions of any under-5 child death on any under-5 sibling death. All regressions include survey indicators and single-year age indicators. Pooled estimations include women of all ages; age-specific estimations are separate for each five-year age group. Average marginal effects are computed for the final age in each age interval; confidence intervals are based on standard errors computed using the delta method. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Figure A.4: Comparison with Other Under-5 Mortality Differentials



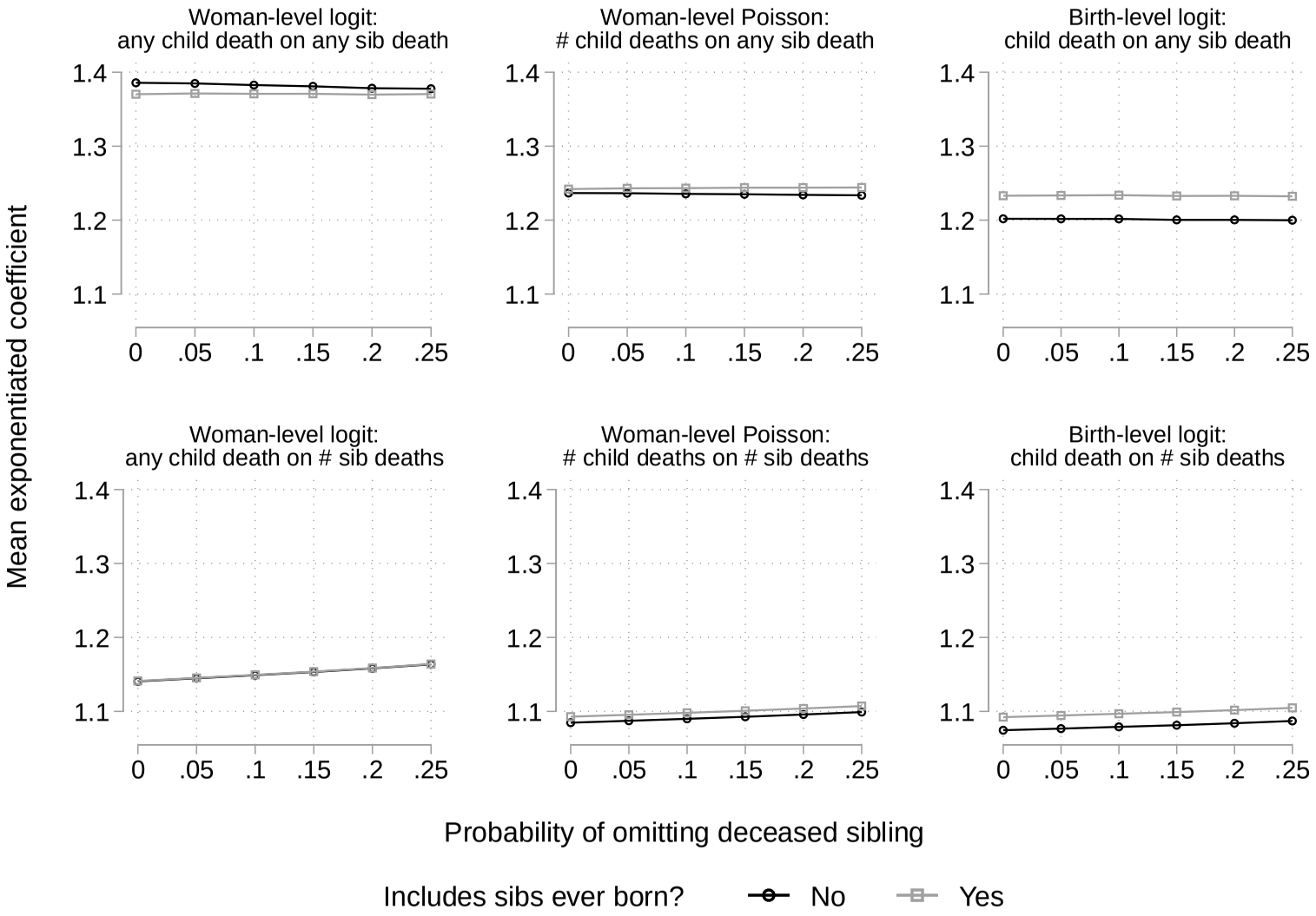
Note: The top panel presents point estimates and 95% confidence intervals of odds ratios from four woman-level logit regressions of any under-5 child death on the indicated categorical variables in the figure. All regressions include survey indicators and single-year age indicators. The bottom panel presents histograms of the categorical variables. Sampling weights are rescaled to reflect each survey's contribution to the sample.

Figure A.5: Robustness to Survey-by-Age Group Effects



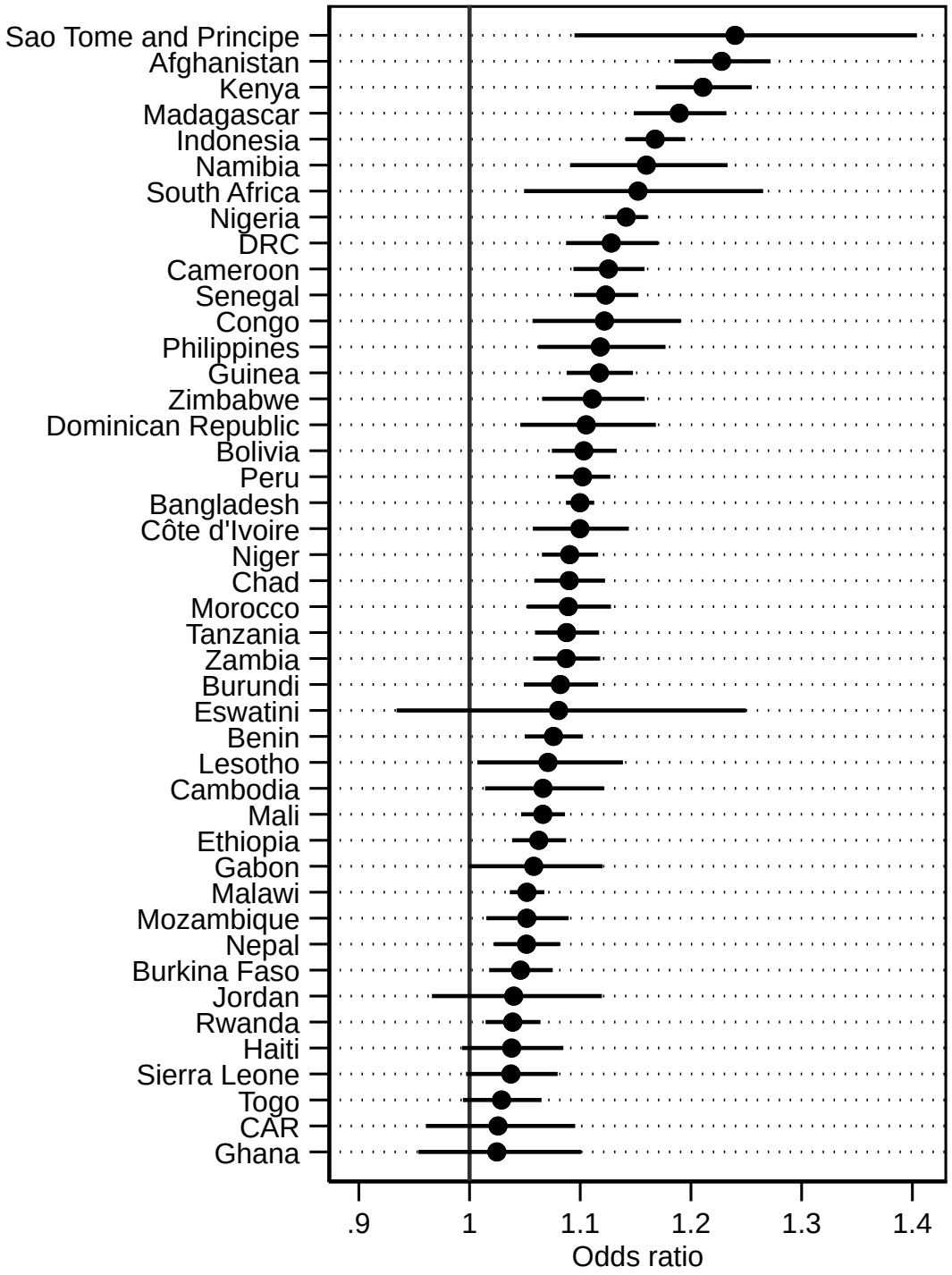
Note: We add survey-by-age group indicators to each regression from Table 2. We report the new estimates alongside the original estimates from Table 2. Spikes represent 95% confidence intervals

Figure A.6: Monte Carlo Simulations of Measurement Error



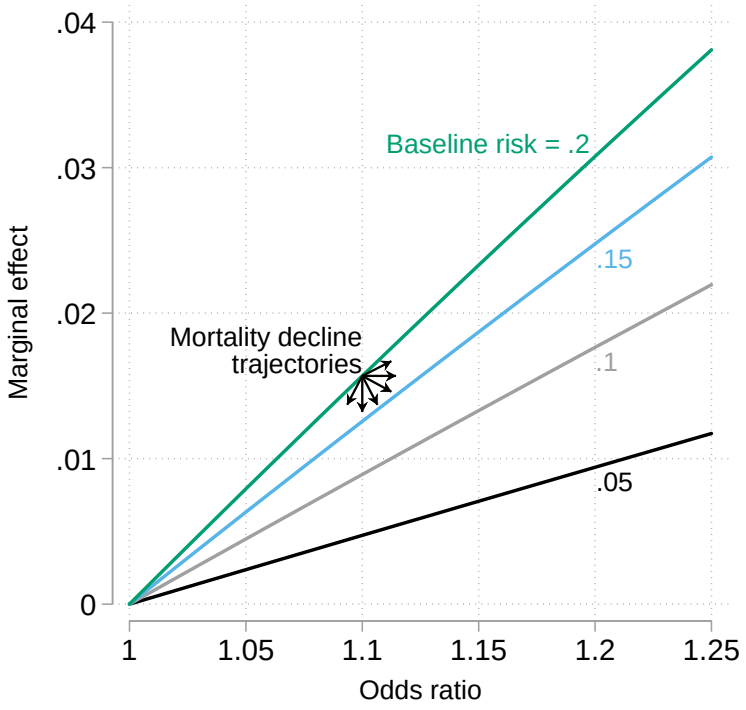
Note: This figure reports the impact of simulated measurement error on our mortality persistence estimates. We simulate the omission of reported deceased siblings for different probabilities of omission. For each positive probability, we draw the number of omitted deceased siblings from a binomial distribution 50 times. We estimate each regression from Table 2 in each simulated dataset. We plot the mean exponentiated coefficient (odds ratio or incidence rate ratio) across the 50 draws. At p = 0, we plot the result from Table 2, with no simulated measurement error.

Figure A.7: Mortality Persistence by Country



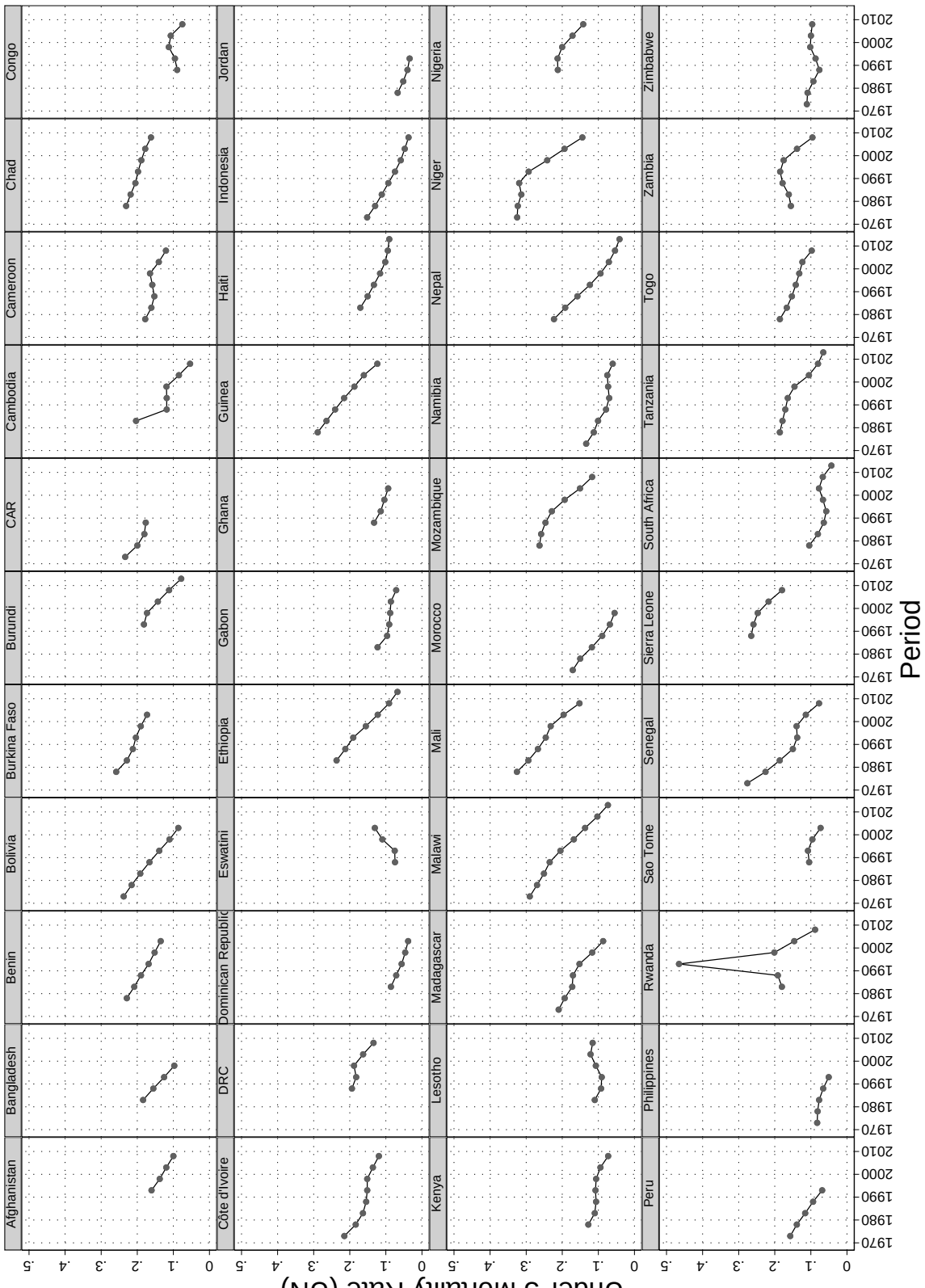
Note: This figure reports mortality persistence estimates for each country in our sample. The plotted estimates are odds ratios from birth-level logit regressions of under-5 death on the mother's number of under-5 sibling deaths. All regressions include the mother's number of siblings ever born and survey indicators. Spikes represent 95% confidence intervals based on standard errors clustered at the survey cluster level. Sampling weights are rescaled to reflect each survey's contribution to each country sample.

Figure A.8: Absolute Versus Proportional Mortality Persistence for a Binary Risk Factor



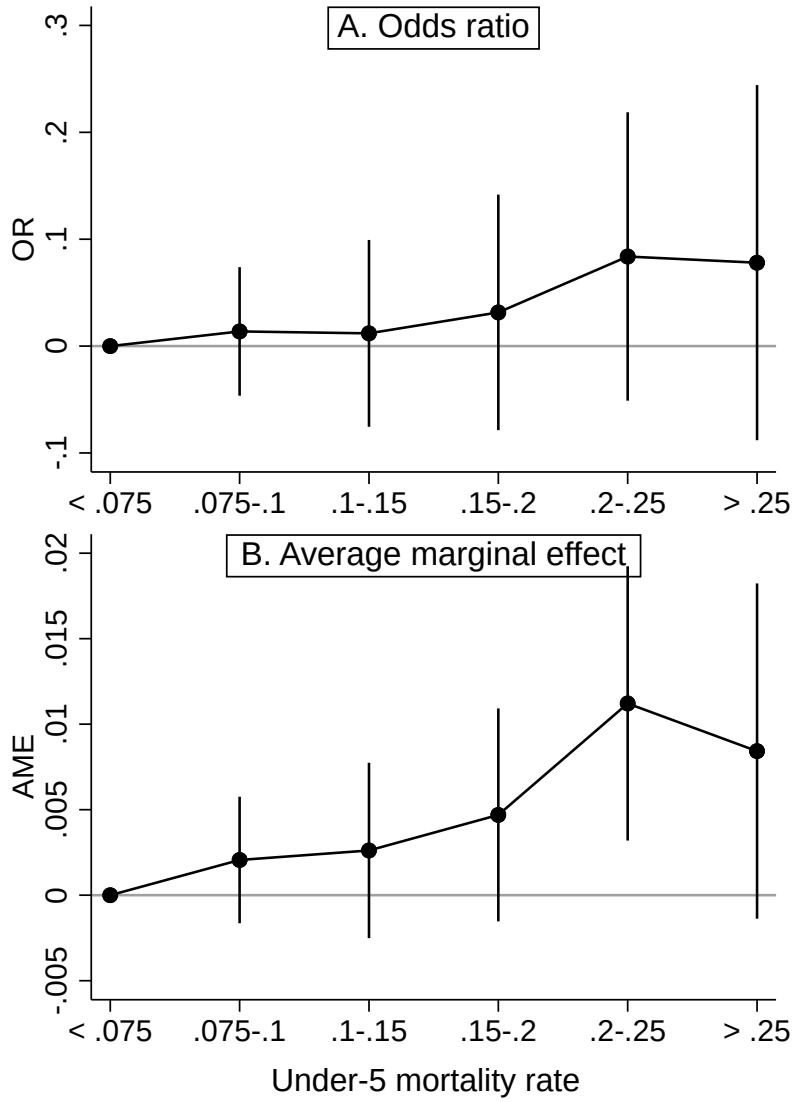
Note: Each ray from the origin specifies the relationship between the marginal effect and the odds ratio for a binary risk factor (e.g., any sibling death) at a given level of baseline mortality risk. At higher baseline mortality risk, a given odds ratio translates to a larger marginal effect. The mortality decline trajectories demonstrate possible paths for the odds ratio and marginal effect as mortality falls.

Figure A.9: Under-5 Mortality Rate over Time, by Country



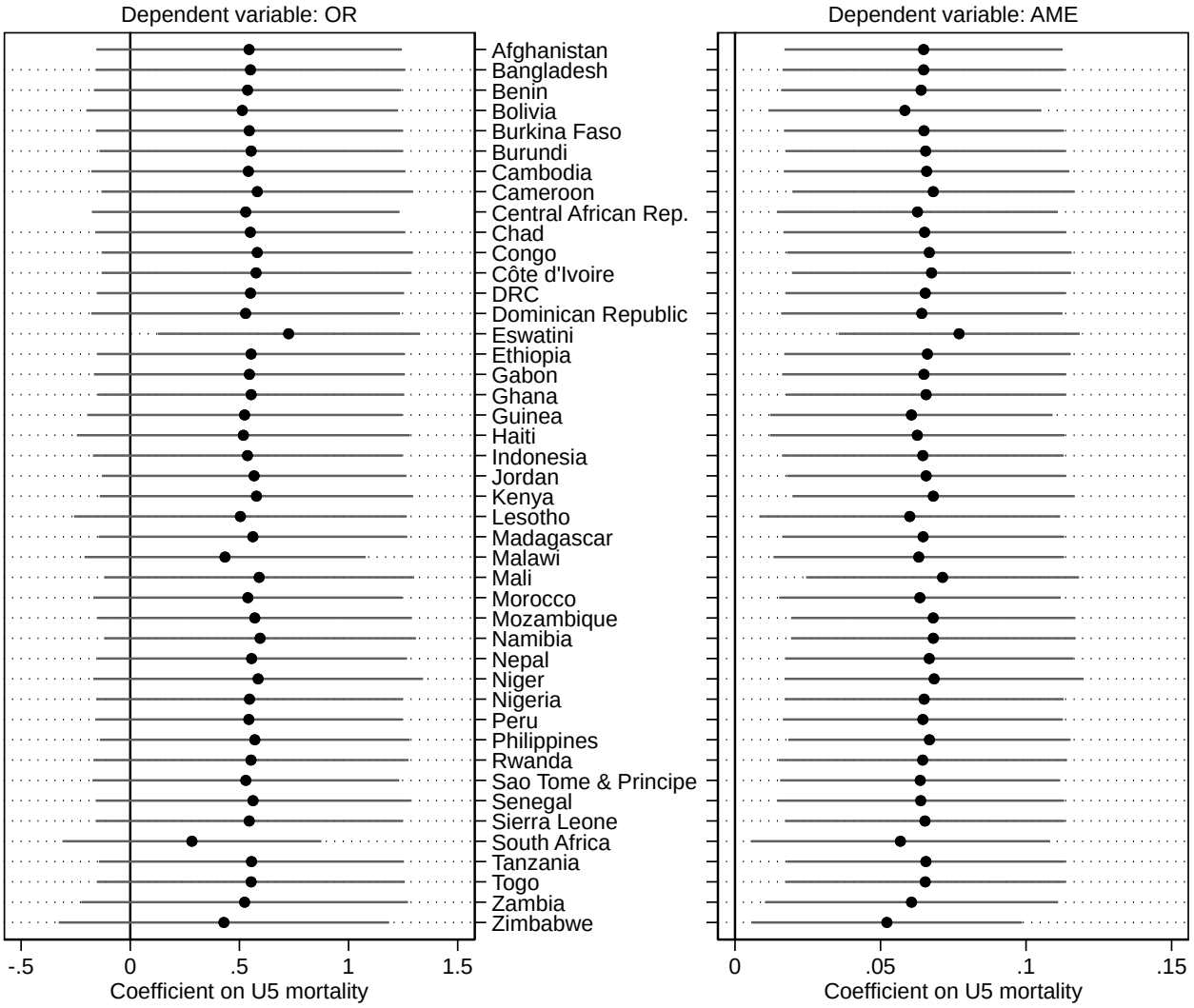
Note: Rates are scaled from 0 to 1.

Figure A.10: Semi-Parametric Panel Analyses



Note: The figure replicates Table 4, columns (1) and (5), but with under-5 mortality separated into 6 bins. The point estimates are the coefficients for 5 bin indicators, leaving out the lowest as the reference category. Spikes are 95% confidence intervals based on standard errors clustered at the country level. OR is the odds ratio. AME is the average marginal effect. Each panel represents a separate cell-level regression including country and period fixed effects. Panel A corresponds to Table 4, column (1), while the Panel B corresponds to Table 4, column (3).

Figure A.11: Leave-One-Out Panel Analyses



Note: This figure replicates Table 4, columns (1) and (5), leaving out one country at a time. The point estimates report the cell-level association of the under-5 mortality rate with the intergenerational persistence of under-5 mortality, net of country fixed effects and period fixed effects. Spikes are 95% confidence intervals based on standard errors clustered at the country level. OR is the odds ratio. AME is the average marginal effect. The left-hand panel corresponds to Table 4, column (1), while the right-hand panel corresponds to Table 4, column (3).