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Reproductive Timing and Family Structure as Determinants of Early-Life Mortality in Historical Poland

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ABSTRACT

This study examines how parental age, birth spacing, sibling number and sex composition, and local socioeconomic and environmental conditions shaped early-life mortality in 19th-century urban Poland. Using reconstructed family histories from the Poznań Historical Population Database, we estimated the risk of infant mortality (before age 1) and child mortality (ages 1–5). Generalized additive models (GAMs) with a binomial logistic link were fitted separately for firstborn (n = 1,023) and laterborn children (n = 2,572). Among firstborns, a larger parental age difference was associated with lower child mortality, potentially reflecting selective mating or differences in parental resources and investment. Among laterborns, infant mortality declined with increasing paternal age, consistent with greater socioeconomic stability among older fathers. Infant mortality was higher following the death of a preceding brother, whereas child mortality was lower among children with a preceding brother. Although birth order and interbirth interval showed no significant direct associations, the protective effect of having a preceding brother weakened with increasing birth order. Mortality risks also varied across parishes, underscoring the importance of local socioeconomic and environmental conditions. Elevated parish-level infant mortality among male firstborns was consistent with established patterns of male vulnerability in early life. Overall, these findings provide new evidence on the determinants of early-life mortality in 19th-century Poznań and highlight the value of integrating biological, familial, and contextual factors when examining early-life survival in historical populations.

Keywords: Infant mortality, Child mortality, Family history, Historical demography, Parental age, Sibling sex composition

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1 INTRODUCTION

According to life history theory, early-life mortality in organisms reflects trade-offs between reproductive timing, parental investment, and offspring viability (Stearns, 2000). In humans, these trade-offs operate both as long-term strategies shaped by early environmental conditions and as shorter-term responses to biological constraints (e.g., maternal age and health), reproductive timing (e.g., birth spacing, birth order, and sibling composition), and current family circumstances (e.g., household resources, family structure, and sibling survival). In historical populations characterized by natural fertility and high mortality, these family-level mechanisms related to reproductive timing and household composition were likely particularly pronounced.

Focusing on proximate demographic and familial mechanisms, this study investigates how variation in parental age, birth spacing, birth order, sibling sex composition, and the vital status of the preceding sibling influenced infant and child mortality in 19th-century urban Poland. Drawing on life history theory and demographic research, the following factors were considered:

Parental age at childbirth, including the age difference between partners (Fieder & Huber, 2007), may influence offspring outcomes through biological, behavioral, and socioeconomic pathways (Goisis et al., 2017). Very young or advanced maternal age is associated with physiological limitations related to pregnancy and childbirth, while advanced paternal age can contribute to genetic and epigenetic risks (Pinheiro et al., 2019; Tarín et al., 1998; Zhang et al., 2022). At the same time, parental experience, stability, and access to household resources often increase with age. Accordingly, higher mortality may be expected among children born to very young or older mothers, and possibly among children of parents with large age differences. However, empirical findings on advanced maternal age are mixed, with evidence of both elevated (Espehaug et al., 1994; Saccone et al., 2022) and reduced infant mortality (Lee et al., 2024), suggesting that these effects are conditioned by broader family and environmental context.

The family environment, including birth spacing, birth order, sibling sex composition, and the vital status of preceding sibling, represents another key determinant of early-life mortality. Short interbirth intervals often reflect high reproductive effort but may also lead to adverse outcomes due to maternal depletion, interrupted breastfeeding, and limited caregiving resources. The vital status of the preceding sibling is also relevant: the early death of a preceding child may shorten subsequent birth intervals through the premature termination of breastfeeding and the associated period of infecundity, thereby increasing both fertility tempo and mortality risk for the next-born child (Fialova et al., 2020). Conversely, very long intervals may indicate delayed reproduction or subfertility. Both very short and very long birth intervals have been linked to low birth weight, undernutrition, and higher neonatal mortality (Miller, 1991; Rutstein, 2005), highlighting the interplay between fertility timing and infant survival. Accordingly, mortality is expected to be higher among children born after very short or very long intervals, as well as among those born following the death of a preceding sibling.

Birth order provides additional insight into familial and biological conditions (Elliott, 1992). While firstborns may experience higher mortality due to reduced maternal physiological efficiency, lower birth weight, and parental inexperience (Bacci et al., 2014; Bohn et al., 2021; Magnus et al., 1985), laterborns may face elevated mortality risks as a result of sibling competition, reduced parental attention, and other challenges associated with larger family size (Bijur et al., 1988; Modin, 2002). Consequently, mortality disadvantages may occur at both ends of the birth-order spectrum, depending on the balance between maternal constraints and parental allocation of resources, potentially interacting with parental age and family composition.

The child's sex and the sex composition of siblings may further interact with the factors described above. Sons and daughters differ biologically in susceptibility to early-life risks through mechanisms such as intrauterine selection, fetal development pace, placental adaptation, and differential resource allocation (Clifton, 2010; Vatten & Skjærven, 2004; Wells, 2000). Social norms may additionally disadvantage daughters through female-selective abortion, intrafamilial neglect, or unequal access to healthcare and nutrition (Beltrán Tapia & Gallego-Martínez, 2020; Lynch, 2011). As a result, higher mortality is predicted for male children, children with a greater number of older brothers, and those born after brothers, reflecting the combined effects of biological vulnerability and sibling competition for resources.

Beyond biological and behavioral mechanisms, early-life mortality is also shaped by broader environmental and temporal factors. Improvements in living conditions, public health, and population

dynamics over time are generally associated with declining mortality, whereas periods of crisis such as epidemics, food shortages, or economic disruption are likely to increase mortality risk. In addition, local variation between parishes may further capture differences in socioeconomic conditions, sanitary infrastructure, population density, and other environmental exposures relevant to child survival (Bradley & Corwyn, 2002). Accordingly, mortality is expected to be higher among children born in poorer parishes or during periods of adverse economic or epidemiological conditions.

2 MATERIALS AND METHODS

The material is drawn from the Poznań Historical Population Database (www.poznandatabase.pl), which contains individual-level information on births, marriages, and deaths recorded in Poznań from the 1830s to the early 20th century. During this period, Poznań, a city in western Poland, was under Prussian administration. Following the partitions of Poland and the resolutions of the Congress of Vienna in 1815, it became the capital of the Grand Duchy of Poznań and subsequently the administrative center of the Poznań Province. Throughout the 19th century, the city functioned as an important administrative, political, and military center under Prussian rule until its incorporation into the re-established Polish state after World War I.

The database covers events recorded across five Catholic parishes (St. Mary Magdalene, St. Margaret, St. Martin, St. Adalbert, and St. John) and one Protestant parish (Holy Cross), as well as Civil Registry Office books introduced in 1874 following Prussian administrative reforms. Using these sources, reproductive histories of individual women were reconstructed by following women born in Poznań until the birth of their own children (n = 3,820). In total, 1,057 completely reconstructed family histories were available and analyzed in this study.

The data included information on the parish in which the marriage was registered, parental birth years and ages at marriage (in full years), the total number of children, and the number of sons and daughters. For each child, the data included the date of birth and death (day, month, year), sex, and birth order. Parental age difference was calculated by subtracting the woman's age at marriage from the man's age at marriage. Parental ages at each birth were calculated by subtracting maternal and paternal birth years from the child's birth year. Child age at death was calculated as the difference between the recorded dates of birth and death, expressed in days, months, and years. The vital status of the preceding sibling was included to indicate whether the previous sibling died before the subsequent birth. To capture the sex composition of previous siblings while avoiding zero counts, smoothed ratios of boys to girls were constructed by adding 0.5 to the number of previous sons and daughters. Interbirth intervals were calculated in days, months, and years between consecutive births for families with two or more children. Families with twin births (n = 114), for which the interbirth interval equals zero, were excluded from the analysis.

To assess the impact of familial characteristics on early-life mortality, we estimated a series of generalized additive models (GAMs) using binomial logistic regression. The outcome variables were binary indicators of infant mortality (death before age 1) and child mortality (death between ages 1 and 5). The age of five was chosen as the upper threshold because deaths up to this age were consistently documented in the material. In contrast, deaths beyond age five were not systematically transcribed from the original records, potentially leading to incomplete mortality information for older children or adults. Separate models were fitted for firstborns and for all laterborn children, i.e., those with birth orders ranging from 2 to 16 (median = 3, mean = 4.06). All models were estimated using the mgcv R-package (Wood, 2017).

For firstborns, we fitted GAMs predicting infant (Model 1) and child (Model 2) mortality as functions of maternal age, paternal age, parental age difference, and birth year, allowing these predictors to enter as smooth terms. Categorical covariates included the child's sex and parish. The total number of firstborns was 1,057, of which 1,023 were included in the models after excluding cases with missing values. Among these, 149 firstborns died during infancy (14.57%), and 105 died between ages 1 and 5 (10.26%).

For laterborn children, we used the bam() function, including a family-level random intercept to account for unobserved heterogeneity and the non-independence of repeated observations within families.

Models predicting infant (Model 3) and child (Model 4) mortality included maternal and paternal ages, maternal age at first birth, parental age difference, birth year, interbirth interval (in months), and the smoothed ratio of boys to girls among previous siblings, with these predictors specified as smooth terms. Categorical covariates included the child's sex, the sex and vital status of the preceding sibling, birth order, and parish. The total number of laterborn children was 2,649, of which 2,572 were included in the models after excluding cases with missing values. Among these, 363 laterborns died during infancy (14.11%), and 289 died between ages 1 and 5 (11.24%).

We first estimated baseline models including main effects, followed by models testing two-way interactions between variables. Only statistically significant interactions ($p < 0.05$) were retained in the final models (i.e., the sex-by-parish interaction in Model 1, the preceding sibling's sex-by-vital status interaction in Model 3, and the preceding sibling's sex-by-birth order interaction in Model 4). Variables with established theoretical relevance (e.g., parental age, birth year) were retained regardless of statistical significance. The basis dimension (k) for each smooth term was determined using gam.check() diagnostics and visual inspection, increasing k when necessary to capture potential nonlinear patterns while avoiding overfitting. Where smooth terms showed no evidence of nonlinearity, estimated effects were effectively linear. Alternative model specifications were compared using the Akaike Information Criterion (AIC) to identify the best-fitting models.

Following the GAM results, we conducted additional tests to evaluate robustness and interpretability. To examine potential non-linear associations, we fitted logistic regression models with quadratic terms for predictors identified as significant in the GAMs. Robustness was then assessed in two ways: first, by bootstrapping the quadratic term (1,000 resamples) to generate empirical confidence intervals; and second, by summarizing raw bin counts of deaths and total children within intervals of the significant predictor to verify that observed patterns were supported by sufficient sample sizes.

Table 1 *Sample distribution and infant and child mortality by parish and sex*

Parish	**Numbers of observations (n)**				**Infant mortality**		**Child mortality**	
	Males	Females	NA	Total	N	%	N	%
Firstborns	525	529	3	1,057	154	14.57	107	10.12
St. Mary Magdalene	111	89	3	203	27	13.30	15	7.39
St. Margaret	251	233		484	106	21.90	75	15.50
St. Martin	45	55		100	8	8.00	4	4.00
Holy Cross	27	35		62	0	0.00	2	3.23
St. Adalbert	39	59		98	10	10.20	4	4.08
St. John	52	58		110	3	2.73	7	6.36
Laterborns	1,363	1,271	15	2,649	380	14.35	297	11.21
St. Mary Magdalene	261	212	8	481	67	13.93	42	8.73
St. Margaret	686	655	7	1,348	265	19.66	224	16.62
St. Martin	110	111		221	17	7.69	6	2.71
Holy Cross	78	66		144	0	0.00	0	0.00
St. Adalbert	197	188		385	29	7.53	23	5.97
St. John	31	39		70	2	2.86	2	2.86
Total	1,888	1,800	18	3,706	534	14.41	404	10.90

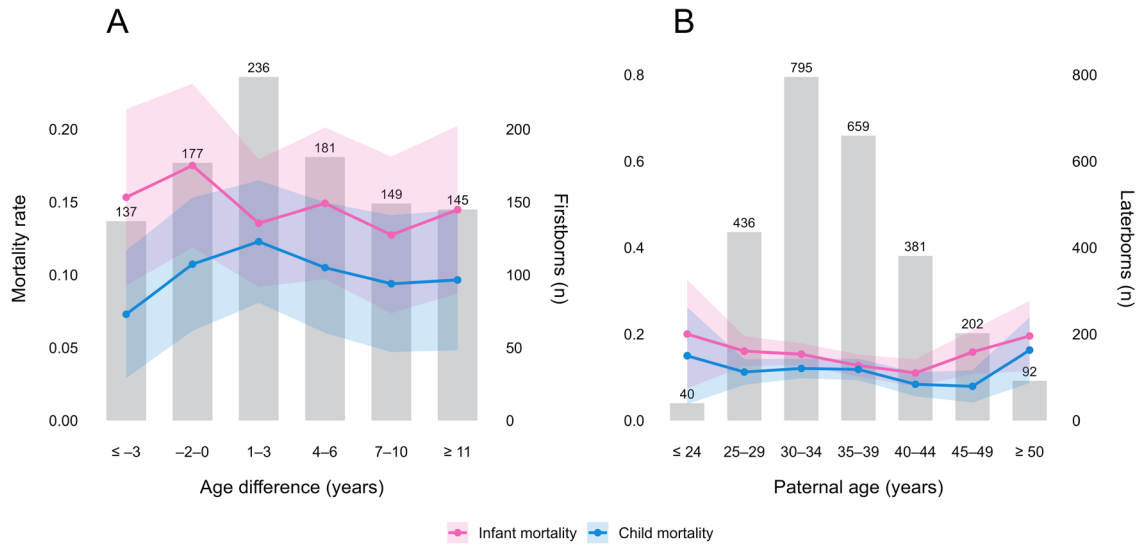
3 RESULTS

In the model predicting infant mortality among firstborns (Model 1), the intercept (–2.706, $p < 0.001$) represents the log-odds of mortality for female infants in the Parish of St. Mary Magdalene (reference parish), indicating a low baseline risk. In this parish, male infants were more likely to die than females ($\beta = 1.179$, $p = 0.026$). Among female infants, mortality was higher in the Parish of St. Margaret compared to the reference parish ($\beta = 1.486$, $p = 0.002$). The interaction between sex and parish indicates that the male–female mortality difference was smaller in the Parish of St. Margaret than in the reference parish ($\beta = -1.265$, $p = 0.028$). For illustration, the model predicts infant mortality of approximately 6.2% for females and 17.8% for males in the Parish of St. Mary Magdalene, whereas in the Parish of St. Margaret predicted mortality was higher among females (about 22.8%) and similar among males (about 21.3%). No significant differences were observed in the other parishes. Maternal age, paternal age, parental age difference, and birth year did not show significant effects and were effectively linear.

For child mortality among firstborns (Model 2), mortality risk was higher in the Parish of St. Margaret compared to the reference parish ($\beta = 0.711$, $p = 0.021$). The child's sex was not a significant predictor ($\beta = -0.116$, $p = 0.586$), and predicted mortality was therefore similar for boys and girls. Birth year showed a nonlinear effect ($\chi^2 = 16.431$, $p = 0.003$), reflecting temporal variation in child mortality. Parental age difference was also associated with child mortality ($\chi^2 = 4.922$, $p = 0.027$), showing an inverted U-shaped pattern (quadratic term = –12, $p = 0.043$). Bootstrap resampling confirmed the robustness of this association, with the quadratic coefficient consistently negative (95% CI –26.0 to –3.9). Descriptive bin counts were consistent with this pattern and showed slightly elevated child mortality at smaller parental age differences (Figure 1, panel A).

Infant mortality among laterborn children (Model 3) showed pronounced parish-level variation: mortality was higher in the Parish of St. Margaret ($\beta = 0.878$, $p < 0.001$) and lower in the Parish of St. John ($\beta = -1.857$, $p = 0.012$) compared to the reference parish. The effect of a preceding male sibling depended on the survival of the previous child: infants whose preceding brother had died faced higher mortality ($\beta = 0.686$, $p = 0.007$). The random intercept for family ID was significant ($\chi^2 = 9.310$, $p = 0.001$), indicating that mortality clustered within families. Paternal age showed a marginally significant association with infant mortality ($\chi^2 = 3.561$, $p = 0.059$), with a significant U-shaped pattern (quadratic term = 7, $p = 0.005$), supported by bootstrapped confidence intervals (95% CI 1.6 to 11.8). Descriptive bin summaries confirmed lowest infant mortality at paternal ages between 41 and 45 years (Figure 1, panel B).

Figure 1 *Significant non-linear predictors of infant and child mortality*



Notes: Panel A shows mortality rates among firstborns by categories of parental age difference, while panel B shows mortality rates among laterborn children by paternal age. Red lines with shaded areas indicate infant mortality rates with 95% confidence intervals, whereas blue lines represent child mortality rates. Gray bars indicate sample sizes (n) for each category among firstborns (panel A) and laterborn children (panel B).

For child mortality among laterborn children (Model 4), risk was again elevated in the Parish of St. Margaret ($\beta = 1.007$, $p < 0.001$) and reduced in the Parish of St. John ($\beta = -1.554$, $p = 0.038$). Having a preceding male sibling was associated with lower mortality overall ($\beta = -0.665$, $p = 0.041$), but this effect varied by birth order ($\beta = 0.144$, $p = 0.027$), indicating that the mortality advantage associated with having a preceding brother diminished with increasing birth order. To further assess this pattern, we also considered a model of overall mortality before age five. In this model, the sex of the preceding sibling showed neither a significant main effect ($\beta = 0.062$, $p = 0.791$) nor an interaction effect ($\beta = 0.020$, $p = 0.676$). Birth year exhibited a strong nonlinear effect ($\chi^2 = 42.862$, $p < 0.001$). The family-level random intercept remained significant ($\chi^2 = 4.898$, $p = 0.013$), reflecting shared unobserved heterogeneity among siblings. Complete GAM results are presented in the Appendix.

4 DISCUSSION

Using reconstructed family histories from 19th-century Poznań, this study first examined associations between parental age characteristics and early-life mortality among their offspring. Despite well-established interest in parental similarity (George et al., 2015; Spuhler, 1968; Watson et al., 2004; Wong et al., 2018), its influence on child outcomes remains unclear. In this study, the only significant association concerned child mortality among firstborns: larger parental age gaps were associated with a lower risk of death before age five. This finding contrasts with expectations that very young or advanced parental ages, or large age differences, would increase mortality risk. Nevertheless, prior research suggests that reproductive success may peak when men are older than their partners (Fieder & Huber, 2007), consistent with evolutionary models emphasizing reproductive potential and resource acquisition (Buss, 1989). Rather than reflecting the independent effects of maternal or paternal age, the protective association of larger age gaps observed here likely reflects the joint configuration of several factors: (1) selective mating, whereby more stable or advantaged couples exhibit greater age differences; (2) higher paternal resources, experience, and social stability associated with older fathers; and (3) increased maternal maturity, caregiving capacity, and accumulated experience among older mothers. Advanced maternal age may also reflect delayed reproduction, leading to increased parental investment in only children or those from smaller families. This interpretation is consistent with the terminal investment hypothesis, which suggests that mothers nearing the end of their reproductive lifespan tend to allocate more resources to offspring (Helle, 2008).

A further consideration is selection through remarriage, as remarried families can exhibit larger parental age gaps and may be positively selected on socioeconomic characteristics, particularly in urban historical contexts. However, the scope for this mechanism is limited in the present study, as most observations involve either first marriages of both spouses or first marriages of women to remarried men. These unions follow similar age-at-marriage patterns, as documented in our previous research (Koníková et al., 2026), and therefore do not introduce substantial differences in parental age gaps. Moreover, the protective association of larger age differences was not evident for infant mortality among firstborns, where an opposite, though statistically non-significant, pattern was observed. This contrast likely reflects different mechanisms: infant mortality is primarily shaped by biological vulnerabilities, including maternal health and perinatal conditions, whereas social, economic, and caregiving factors may play a larger role during childhood. The association was also absent among laterborn children, suggesting that benefits associated with parental age differences are most relevant during early stages of family formation.

Paternal age was associated with infant mortality among laterborn children, with mortality declining as paternal age increased until approximately 45 years, after which mortality risk rose. This finding may suggest that the association between paternal age and child survival likely reflects socioeconomic advantages and accumulated resources. In our previous work, fathers in higher-status occupations tended to be older (Koníková et al., 2026). In the high-mortality context of past societies, where the economic burden of raising multiple offspring was substantial, the availability of resources may have been particularly important for child survival (Hacker et al., 2023; Jaadla et al., 2020). In contrast, maternal age effects were not observed in any of the analyses, suggesting that maternal contributions were essential across all ages and that mortality risk therefore varied less by maternal age.

Second, early-life mortality was examined in relation to the child's sex and family environment, including birth order, birth spacing, the sex and vital status of the preceding sibling, and the ratio of boys to girls among previous siblings. Among firstborns, male infants in the Parish of St. Mary Magdalene faced a higher risk of mortality in the first year of life than females, consistent with evidence of stronger intrauterine selection and greater male vulnerability to adverse outcomes such as neonatal morbidity and mortality (Clifton, 2010; Vatten & Skjærven, 2004). Beyond infancy, and among laterborn children, sex differences were not evident, indicating that male disadvantage was most pronounced during the infancy period of first pregnancies.

Among laterborn children, the association between a preceding male sibling and infant mortality depended on the survival of the previous child: infants whose preceding brother had died experienced higher mortality. Following a more physiologically demanding male pregnancy (Di Renzo et al., 2007), the loss of a preceding son may indicate heightened maternal depletion or stress, or shared adverse conditions, increasing vulnerability in the next offspring. In contrast, having a preceding male sibling was associated with lower child mortality, possibly because the most vulnerable children had already died during infancy. This protective effect weakened with increasing birth order, suggesting cumulative maternal strain from successive demanding pregnancies and intensified sibling competition for parental resources. Previous research similarly indicates that boys with more same-sex siblings face reduced survival chances after age one, though not in infancy (Riswick, 2018).

Contrary to previous findings linking higher birth order (Modin, 2002) and suboptimal interbirth intervals (Miller, 1991; Rutstein, 2005) with increased early-life mortality, no significant effects of either factor were observed in this study. As noted by Elliott (1992), birth order may function more as a proxy for underlying familial or social conditions than as an independent predictor of child health outcomes.

Third, mortality patterns reflected local socioeconomic and demographic conditions, as indicated by birth year and parish. The Catholic Parish of St. Margaret consistently showed the highest mortality risk, whereas St. John Parish was associated with lower mortality. Although both parishes were impoverished (Liczbińska, 2009), the rural character of St. John and its location on the outskirts of the city may have provided a more favorable environment. Elevated mortality in the Parish of St. Margaret aligns with previous research linking poverty, poor sanitation, and limited access to healthcare to adverse child outcomes (Bradley & Corwyn, 2002; Letourneau et al., 2013). It should be noted that estimates for Holy Cross Parish were imprecise due to the low number of observed infant and child deaths in this parish, likely reflecting limited sample size rather than meaningful differences in underlying mortality risk.

While birth year was not significantly associated with infant mortality, it showed a nonlinear relationship with child mortality among both firstborn and laterborn children. This pattern likely reflects historical fluctuations in mortality driven by epidemic outbreaks (e.g., cholera in 1831, 1837, 1848, 1852, 1855, 1866, 1873; scarlet fever and typhus in 1863), followed by a general decline in mortality over time, linked to improvements in urban public health, sanitation, and social infrastructure.

5 RESEARCH LIMITATIONS

Several limitations should be noted. First, mortality estimates are based on historical parish and civil records, which may contain inaccuracies, incomplete entries, or underreporting. In addition, as with all family reconstruction studies, the analyses are limited to individuals who could be followed within the same parish over time. Given the high levels of migration and residential mobility in 19th-century Poznań, only a small subset of families could be reconstructed across the full reproductive life course. This restriction likely favors individuals with greater residential stability and potentially more favorable social or health conditions. Consequently, the absolute levels of child mortality observed in this study should not be interpreted as representative of the broader urban population. Rather, the results primarily reflect associations between child mortality and parental characteristics within families for whom complete life-course data are available.

Second, although generalized additive models (GAMs) allowed us to capture nonlinear effects and interactions, residual confounding may persist due to unmeasured family- or community-level factors, such as parental health, maternal nutrition, household conditions, or access to local resources. Most importantly, we are unable to include information on parental loss, a key determinant of child mortality.

In our data, reconstructed family histories are limited to the reproductive life course of women, and later-life mortality is not consistently documented. Consequently, we cannot distinguish true survival from missing information, which prevents the construction of a reliable measure of parental loss within the required time frame.

Third, the observed effects of parental age, although statistically significant, were modest and may reflect context-specific patterns in urban 19th-century Poznań, including local socioeconomic conditions, family size norms, and demographic structure. Caution is therefore warranted when generalizing these findings to other populations or historical periods. Future research should replicate these analyses in larger and less selectively constrained datasets, incorporate more detailed socioeconomic and environmental variables, and explicitly address migration and parental survival to further assess the robustness and generalizability of the observed patterns.

6 CONCLUSION

This study is among the first to utilize reconstructed family histories from the Poznań Historical Population Database, providing initial insights into the determinants of early-life mortality in a historical urban setting. We found that larger parental age differences were associated with improved child survival among firstborns, while advancing paternal age (up to 45 years) was linked to lower infant mortality among laterborn children. These patterns appear to reflect social and economic advantages rather than purely biological effects. Apart from sex-based mortality differences, which were most pronounced during infancy, classic predictors such as birth order and interbirth interval showed no significant effects, suggesting that their influence may depend on broader familial or social contexts. Socioeconomic and environmental factors, particularly parish-level disparities, substantially shaped mortality risks across all children. These exploratory findings underscore the importance of integrating biological, behavioral, and familial factors when examining early-life survival in historical populations.

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DATA AVAILABILITY

The data that supports the findings of this study are available from the authors upon reasonable request.

REFERENCES

Bacci, S., Bartolucci, F., Chiavarini, M., Minelli, L., & Pieroni, L. (2014). Differences in birthweight outcomes: A longitudinal study based on siblings. *International Journal of Environmental Research and Public Health*, *11*(6), 6472–6484. https://doi.org/10.3390/ijerph110606472

Beltrán Tapia, F. J., & Gallego-Martínez, D. (2020). What explains the missing girls in nineteenth-century Spain? *The Economic History Review*, *73*(1), 59–77. https://doi.org/10.1111/ehr.12772

Bijur, P. E., Golding, J., & Kurzon, M. (1988). Childhood accidents, family size and birth order. *Social Science & Medicine*, *26*(8), 839–843. https://doi.org/10.1016/0277-9536(88)90176-1

Bohn, C., Vogel, M., Poulain, T., Spielau, U., Hilbert, C., Kiess, W., & Körner, A. (2021). Birth weight increases with birth order despite decreasing maternal pregnancy weight gain. *Acta Paediatrica*, *110*(4), 1218–1224. https://doi.org/10.1111/apa.15598

Bradley, R., & Corwyn, R. (2002). Socioeconomic status and child development. *Annual Review of Psychology*, *53*, 371–399. https://doi.org/10.1146/annurev.psych.53.100901.135233

Buss, D. M. (1989). Sex differences in human mate preferences: Evolutionary hypotheses tested in 37 cultures. *Behavioral and Brain Sciences*, *12*(1), 1–14. https://doi.org/10.1017/S0140525X00023992

Clifton, V. L. (2010). Review: Sex and the human placenta: Mediating differential strategies of fetal growth and survival. *Placenta*, *31*(Suppl), S33–S39. https://doi.org/10.1016/j.placenta.2009.11.010

Di Renzo, G. C., Rosati, A., Sarti, R. D., Cruciani, L., & Cutuli, A. M. (2007). Does fetal sex affect pregnancy outcome? *Gender Medicine*, *4*(1), 19–30. https://doi.org/10.1016/S1550-8579(07)80004-0

Elliott, B. A. (1992). Birth order and health: Major issues. *Social Science & Medicine*, *35*(4), 443–452. https://doi.org/10.1016/0277-9536(92)90337-p

Espehaug, B., Daltveit, A. K., Vollset, S. E., Oyen, N., Ericson, A., & Irgens, L. M. (1994). Infant survival in Norway and Sweden 1985–88. *Acta Paediatrica*, *83*(9), 977–982. https://doi.org/10.1111/j.1651-2227.1994.tb13186.x

Fialova, L., Hulikova Tesarkova, K., & Janakova Kuprova, B. (2020). The 'high infant mortality trap': The relationship between birth intervals and infant mortality — The example of two localities in Bohemia between the 17th and 19th centuries. *The History of the Family, 25*(1), 94–134. https://doi.org/10.1080/1081602X.2019.1650792

Fieder, M., & Huber, S. (2007). Parental age difference and offspring count in humans. *Biology Letters, 3*(6), 689–692. https://doi.org/10.1098/rsbl.2007.0324

George, D., Luo, S., Webb, J., Pugh, J., Martinez, A., & Foulston, J. (2015). Couple similarity on stimulus characteristics and marital satisfaction. *Personality and Individual Differences*, *86*, 126–131. https://doi.org/10.1016/j.paid.2015.06.005

Goisis, A., Schneider, D. C., & Myrskylä, M. (2017). The reversing association between advanced maternal age and child cognitive ability: Evidence from three UK birth cohorts. *International Journal of Epidemiology*, *46*(3), 850–859. https://doi.org/10.1093/ije/dyw354

Hacker, J. D., Dribe, M., & Helgertz, J. (2023). Wealth and child mortality in the nineteenth-century United States: Evidence from three panels of American couples, 1850–1880. *Social Science History*, *47*(3), 333–366. https://doi.org/10.1017/ssh.2023.12

Helle, S. (2008). Why twin pregnancies are more successful at advanced than young maternal age? A potential role of 'terminal reproductive investment'. *Human Reproduction*, *23*(10), 2387–2389. https://doi.org/10.1093/humrep/den305

Jaadla, H., Potter, E., Keibek, S., & Davenport, R. (2020). Infant and child mortality by socio-economic status in early nineteenth-century England. *The Economic History Review*, *73*(4), 991–1022. https://doi.org/10.1111/ehr.12971

Koníková, L., Liczbińska, G., & Králík, M. (2026). Marital choices in the 19th-century Poznań: Interplay of origin, occupation, and age at marriage. *Human Nature, 36*, 588–608. https://doi.org/10.1007/s12110-026-09511-6

Lee, D. S., Barclay, K. J., Magnus, M. C., Ernst, A., & Myrskylä, M. (2024). *Multiple births mortality by maternal age at birth: A within-family analysis of Demographic and Health Survey data on 42 low-income countries* (Working Paper WP-2024-034). Max Planck Institute for Demographic Research. https://doi.org/10.4054/MPIDR-WP-2024-034

Letourneau, N. L., Duffett-Leger, L., Levac, L., Watson, B., & Young, C. (2013). Socioeconomic status and child development: A meta-analysis. *Journal of Emotional and Behavioral Disorders*, *21*(3), 211–224. https://doi.org/10.1177/1063426611421007

Liczbińska, G. (2009). Infant and child mortality among Catholics and Lutherans in nineteenth century Poznań. *Journal of Biosocial Science*, *41*(5), 661–683. https://doi.org/10.1017/S0021932009990101

Lynch, K. A. (2011). Why weren't (many) European women 'missing'? *The History of the Family*, *16*(3), 250–266. https://doi.org/10.1016/j.hisfam.2011.02.001

Magnus, P., Berg, K., & Bjérkedal, T. (1985). The association of parity and birth weight: Testing the sensitization hypothesis. *Early Human Development*, *12*(1), 49–54. https://doi.org/10.1016/0378-3782(85)90136-7

Miller, J. E. (1991). Birth intervals and perinatal health: An investigation of three hypotheses. *Family Planning Perspectives*, *23*(2), 62–70. https://doi.org/10.2307/2135451

Modin, B. (2002). Birth order and mortality: A life-long follow-up of 14,200 boys and girls born in early 20th century Sweden. *Social Science & Medicine*, *54*(7), 1051–1064. https://doi.org/10.1016/S0277-9536(01)00080-6

Pinheiro, R. L., Areia, A. L., Mota Pinto, A., & Donato, H. (2019). Advanced maternal age: Adverse outcomes of pregnancy, a meta-analysis. *Acta Médica Portuguesa, 32*(3), 219–226. https://doi.org/10.20344/amp.11057

Riswick, T. (2018). Testing the conditional resource-dilution hypothesis: The impact of sibship size and composition on infant and child mortality in the Netherlands, 1863–1910. *The History of the Family*, *23*(4), 623–655. https://doi.org/10.1080/1081602X.2018.1532310

Rutstein, S. O. (2005). Effects of preceding birth intervals on neonatal, infant and under-five years mortality and nutritional status in developing countries: Evidence from the demographic and health surveys. *International Journal of Gynaecology & Obstetrics*, *89*(Suppl 1), S7–S24. https://doi.org/10.1016/j.ijgo.2004.11.012

Saccone, G., Gragnano, E., Ilardi, B., Marrone, V., Strina, I., Venturella, R., Berghella, V., & Zullo, F. (2022). Maternal and perinatal complications according to maternal age: A systematic review and meta-analysis. *International Journal of Gynecology & Obstetrics*, *159*(1), 43–55. https://doi.org/10.1002/ijgo.14100

Spuhler, J. N. (1968). Assortative mating with respect to physical characteristics. *Eugenics Quarterly*, *15*(2), 128–140. https://doi.org/10.1080/19485565.1968.9987763

Stearns, S. C. (2000). Life history evolution: Successes, limitations, and prospects. *Naturwissenschaften*, *87*, 476–486. https://doi.org/10.1007/s001140050763

Tarín, J. J., Brines, J., & Cano, A. (1998). Long-term effects of delayed parenthood. *Human Reproduction, 13*(9), 2371–2376. https://doi.org/10.1093/humrep/13.9.2371

Vatten, L. J., & Skjærven, R. (2004). Offspring sex and pregnancy outcome by length of gestation. *Early Human Development*, *76*(1), 47–54. https://doi.org/10.1016/j.earlhumdev.2003.10.006

Watson, D., Klohnen, E. C., Casillas, A., Simms, N., Haig, J., & Berry, D. S. (2004). Match makers and deal breakers: Analyses of assortative mating in newlywed couples. *Journal of Personality, 72*(5), 1029–1068. https://doi.org/10.1111/j.0022-3506.2004.00289.x

Wells, J. C. (2000). Natural selection and sex differences in morbidity and mortality in early life. *Journal of Theoretical Biology*, *202*(1), 65–76. https://doi.org/10.1006/jtbi.1999.1044

Wong, Y. K., Wong, W. W., Lui, K. F. H., & Wong, A. C.-N. (2018). Revisiting facial resemblance in couples. *PLOS ONE*, *13*(1), e0191456. https://doi.org/10.1371/journal.pone.0191456

Wood, S. N. (2017). *Generalized additive models: An introduction with R* (2nd ed.). Chapman & Hall CRC

Zhang, C., Yan, L., & Qiao, J. (2022). Effect of advanced parental age on pregnancy outcome and offspring health. *Journal of Assisted Reproduction and Genetics, 39*(9), 1969–1986. https://doi.org/10.1007/s10815-022-02533-w

APPENDIX

Table A1 *Summaries of generalized additive models predicting infant mortality (before age 1) and child mortality (ages 1–5) among firstborn (n = 1,023) and later-born children (n = 2,572). Significance codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '·' 0.1 ' ' 1*

Firstborns	**Infant mortality (Model 1)**				**Child mortality (Model 2)**			
Parametric coefficients	Estimate	Standard error	Z value	P value	Estimate	Standard error	Z value	P value
(Intercept)	-2.706	0.463	-5.839	0.000 ***	-2.494	0.304	-8.210	0.000 ***
Parish of St. Margaret	1.486	0.491	3.027	0.002 **	0.711	0.307	2.318	0.021 *
Parish of St. Martin	0.396	0.666	0.594	0.552	-0.852	0.592	-1.440	0.150
Parish of Holy Cross	-39.890	11,340,000	0.000	1.000	-1.028	0.782	-1.314	0.189
Parish of St. Adalbert	-0.283	0.764	-0.371	0.711	-0.792	0.607	-1.306	0.192
Parish of St. John	-1.278	1.111	-1.151	0.250	-0.376	0.485	-0.776	0.438
Male sex	1.179	0.530	2.224	0.026 *	-0.116	0.213	-0.544	0.586
Male sex: St. Margaret	-1.265	0.575	-2.198	0.028 *				
Male sex: St. Martin	-1.523	0.927	-1.643	0.100				
Male sex: Holy Cross	-1.106	17,190,000	0.000	1.000				
Male sex: St. Adalbert	0.208	0.898	0.232	0.817				
Male sex: St. John	-0.427	1.349	-0.317	0.752				

Smooth terms	Estimated df	Reference df	Chi-square	P value	Estimated df	Reference df	Chi-square	P value
Maternal age	1.000	1.000	0.603	0.437	1.000	1.000	1.289	0.256
Paternal age	1.000	1.000	1.361	0.243	1.000	1.001	0.996	0.318
Age difference	0.000	0.000	0.000	0.998	0.843	0.975	4.922	0.027 *
Birth year	1.000	1.000	0.033	0.855	3.737	4.158	16.431	0.003 **

Laterborns	**Infant mortality (Model 3)**					**Child mortality (Model 4)**				
Parametric coefficients	Estimate	Standard error	Z value	P value		Estimate	Standard error	Z value	P value	
(Intercept)	-1.978	0.350	-5.650	0.000	***	-1.783	0.423	-4.215	0.000	***
Parish of St. Margaret	0.878	0.228	3.858	0.000	***	1.007	0.249	4.042	0.000	***
Parish of St. Martin	0.349	0.434	0.804	0.421		-0.820	0.549	-1.493	0.136	
Parish of Holy Cross	-12.356	120.6	-0.102	0.918		-12.299	111.334	-0.110	0.912	
Parish of St. Adalbert	0.392	0.436	0.898	0.369		0.054	0.456	0.118	0.906	
Parish of St. John	-1.857	0.742	-2.504	0.012	*	-1.554	0.749	-2.074	0.038	*
Male sex	0.123	0.117	1.049	0.294		-0.134	0.130	-1.036	0.300	
Birth order	0.048	0.066	0.729	0.466		-0.060	0.085	-0.711	0.477	
Previous male sex	0.024	0.165	0.145	0.885		-0.665	0.324	-2.049	0.041	*
Previous male sex: order						0.144	0.065	2.219	0.027	*
Previous sibling death	0.141	0.195	0.724	0.469		0.014	0.148	0.097	0.923	
Previous male sex: death	0.686	0.254	2.696	0.007	**					

Smooth terms	Estimated df	Reference df	Chi-square	P value		Estimated df	Reference df	Chi-square	P value	
Maternal age	1.000	1.000	0.039	0.843		1.000	1.000	0.392	0.531	
Maternal age at first birth	1.000	1.000	0.056	0.813		1.000	1.000	0.652	0.420	
Paternal age	0.781	0.951	3.561	0.059	.	0.000	0.000	0.000	0.500	
Age difference	1.000	1.000	0.515	0.473		1.000	1.000	0.657	0.418	
Interbirth interval	1.000	1.000	1.351	0.245		1.000	1.000	0.194	0.659	
Previous sex ratio	1.000	1.000	0.088	0.766		1.000	1.000	0.647	0.421	
Birth year	2.035	2.536	3.257	0.339		4.558	4.883	42.862	0.000	***
Family ID	0.903	1.000	9.310	0.001	**	0.838	1.000	4.898	0.013	*