J-Curve?: A Meta-Analysis of the Association between Parity and All-Cause Parental Mortality

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ABSTRACT

Previous studies have shown a relationship between parity and associated parental health and mortality. By and large, the extant literature has credited either low parity or high parity with influencing the long-term health or risk of mortality of parents. Some studies, however, have shown that both low and high mortality influences the risk of all-cause parental mortality. Overall, however, the extent to which parity is associated with parental mortality is inconclusive in the extant literature. We extracted 165 independent measures of relative mortality risks from 30 studies, both historical and contemporary, to examine whether the J-curve association found in a few studies held across many studies. The results of our meta-analyses and meta-regressions suggest that there is indeed a J-curve association between parity and all-cause parental mortality such that both low parity and high parity increase the risk of all-cause parental mortality across a range of national contexts.

Introduction

Social scientists have long considered important the influence of childbearing and rearing on long-term health outcomes for parents and children. In particular, interest in childbearing has increased due to changes in childbearing patterns, including parity, over the past few decades. Many couples today delay childbearing in pursuit of education and career opportunities, many have fewer children than in prior decades, and other couples opt out of childbearing entirely (Martin 2004). Some scholars argue that childbearing and rearing is an integral part of the life course, wherein parents' and children's lives are linked and interdependent (Elder 1998) and affect one another throughout the life course (e.g., Umberson, Purdrovska, and Peczek 2010). Intuitively, the extent to which parents' and children's 'linked lives' affect parental (and offspring) outcomes should be, at least in part, associated with the number of children they bear and rear (i.e., parity).

Indeed, the extant literature suggests that parity is associated with all-cause parental mortality, although some studies suggest that the association is complex. That is, parity tends to predict all-cause parental mortality (e.g., Penn and Smith 2007), but some studies suggest that the relationship may be nonlinear. That is, the relationship between parity and parental mortality may form a U- or J-shaped curve (Dior et al. 2013; Doblhammer, 2000; Green et al. 1988; Högberg and Wall, 1986; Jaffe et al. 2009) wherein parity predicts increased mortality for those with low (or nulliparous) and high parity but not for those with moderate parity. The extent to which the relationship between parity and all-cause parental mortality forms a J-shaped curve is important to understand given the significant changes in families and childbearing over the last half century. Yet, only a few studies in a large body of literature have directly examined whether the association between parity and mortality is nonlinear or forms a J-shaped curve.

This paper extends the extant literature by using meta-analytic and meta-regression techniques to examine the hypothesis that the association between parity and all-cause mortality is nonlinear or forms a J-shaped curve. In addition, because of the potential toll of childbearing on women's versus men's bodies and potential differences in their health and longevity, we further examine gender as a possible moderator. Unlike prior studies, which focus on a specific country or a comparison between two countries, our data include multiple national contexts from both historical and contemporary studies ($N = 30$) and span a range of disciplines, theoretical perspectives, and methodological approaches. In addition, our meta-analytic approach allows us to extend the literature by using results from existing studies (including those which do not directly examine a J-curve association) to more extensively examine the nonlinear or J-curve hypothesis. Meta-analytic and meta-regression results suggest that there is a significant nonlinear association between parity and all-cause parental mortality. Unexpectedly, our results further suggest that gender does not moderate the association between parity and all-cause parental mortality.

Theory and Background

Scholarship focused on the association between parity and parental mortality spans a range of disciplinary perspectives and methodological approaches. We focus our review on evolutionary/biological, biomedical, and social perspectives with an emphasis on the J-curve association between parity and all-cause parental health and mortality found in the extant literature. We further highlight potential differences in the association between parity and mortality for men versus women, and finally, we review possible sources of social selection which may explain the nonlinear association between parity and all-cause parental mortality.

Evolutionary and Biological Perspectives

Evolutionary and biological theoretical perspectives emphasize the physical repercussions of pregnancy and childrearing. The general idea is that childbearing may result in premature degenerative health; some scholars argue that there is a physical downside to pregnancy and childbirth which may shorten the life of mothers. Theories such as disposable soma (Alter, Dribe, and Van Poppel 2007; Dribe 2007) and the evolution of senescence (Doblhammer 2000; Hurt, et al. 2006) consider the metabolic and physiological trade-offs between parity and mortality. Disposable soma theory (and senescence) posits that those with higher parity invest more in fertility than they do in biological resources which may be otherwise used for bodily maintenance and cell reparation; thus, higher parity accelerates the aging process (see Le Bourg 2001). On the other hand, some scholars suggest that these evolutionary processes may depend on the timing of childbirth (Alter, Dribe, and Van Poppel 2007); early childbearing may increase the risk of health problems and decrease longevity.

Evolutionary/biological theories often focus on non-human populations in which maximizing fitness through fertility is more important than longevity, and in which the social and cultural complexities underlying human life are not a consideration (Drive 2004). Moreover, disposable soma implies that one either invests in fertility or longevity, each at the expense of the other, but evidence on human populations suggests that at least some investment in fertility increases longevity among humans. Read, Grundy, and Wolf (2011), for example, find that nulliparous women are at a greater risk of becoming inactive as a result of poor health compared to parous women. Among parous adults, Read and colleagues further found that both men and women who have higher versus lower parity are at an increased risk of poor health. This nonlinear association (among other findings) between parity and longevity suggests that the

evolutionary trade-off between fertility and longevity may be less relevant in human versus non-human populations.

Biomedical Perspectives

Biomedical theories, much like evolutionary/biological perspectives, emphasize the physical links between parity, health, and mortality. These theories posit that the onset of chronic diseases is, at least in part, a function of parity (e.g., Alter et al. 2007; Read, Grundy, and Wolf 2011). In particular, the hormonal fluctuations that mothers experience during gestation, delivery, and lactation may be associated with life-threatening conditions such as cancer and cardiovascular disease (Daling et al. 2002; Hurt et al. 2006; Alter et al. 2007). Increased exposure to hormonal fluctuations, with increasing parity, conceivably increases the risk of disease onset. Henretta (2007) found that hormonal changes which occur during both pregnancy and childbirth increase susceptibility to diabetes, heart disease, and cancer, all of which increase the risk of mortality. Similarly, giving birth within two years preceding a breast cancer diagnosis significantly increases hormonal fluctuations during pregnancy and childbirth and the risk of post-reproductive mortality (Daling et al. 2002). Additional research suggests that parity increases susceptibility to infections, and parity has also been linked to depression (Grundy and Kravdal 2008).

Physiological and emotional stress associated with multiple pregnancies, in addition to the economic strain of rearing multiple children, may take a physical toll on the body and lead to poorer health and mortality. Parity in and of itself may be a source of stress, as greater numbers of children may require increased emotional, physical, and financial investments. Maternal depletion models, for example, suggest that good nutrition is harder to come by among women with higher parity; higher parity women may trade in their own health for the health of their

children due to resource depletion; shorter durations between multiple childbirths may increase stress; and higher parity may increase exposure to stress and the subsequent contraction of diseases (see Alter, Dribe, and Van Poppel 2007).

Notwithstanding the health risks associated with multiple pregnancies, numerous studies (e.g., Alter et al. 2007; Dribe 2007; Grundy and Kravdal, 2008; Jaffe et al. 2009) note that the physiological risk factors associated with higher parity are exacerbated among parents who have few resources and live in lower versus higher socioeconomic environments. Lower versus higher SES parents may be less able to purchase nutritious food during and following pregnancy and may have less access to stress-reducing resources (e.g., time to exercise). Overall, lower versus higher socioeconomic families are at an increased risk of experiencing depression and related illness (Lorant et al. 2003). Even so, using census-based data from the Israel Longitudinal Mortality Study, Jaffe et al. (2009) found a significant, nonlinear association between parity and all-cause mortality among both men and women even after controlling for socioeconomic status and other demographic characteristics such as age and marital status. Jaffe and colleagues argue that the association between parity and all-cause mortality is not likely linked to pregnancy but rather is "likely mediated by biological and psychological factors and other lifestyle characteristics that have long-term consequences into older ages" (Jaffe et al. 2009: 9). We concur and argue that the social contexts in which parents and children are embedded, and from which their lifestyle characteristics emerge, are also important for understanding the nonlinear association between parity and all-cause parental mortality.

Social Perspectives

Sociological life course perspectives underscore the collective experiences of parents and children over time and the potential consequences of those experiences (e.g., Elder 1998). As we

noted, childbearing and parity in particular, influence parental longevity and mortality (e.g., Dior et al. 2013; Doblhammer 2000; Jaffe et al. 2009; Read, Grundy, & Wolf 2011). The mere presence of children, for example, may be a form of social control for parents. Conceivably, parents versus nonparents take fewer health risks such as abusing drugs and alcohol (e.g., Umberson 1987). Of course, people who have children may simply make better health decisions than those who do not (i.e., selection), but the transition to parenthood may also reduce risky health behaviors because parents want to protect their children (e.g., against exposure to second-hand smoke) and remain healthy well into their children's adulthood. While adults may make healthier decisions once they become parents (i.e., nulliparous adults may live less healthy lives), exercise and/or buying healthy food (which tends to be more expensive than unhealthy food) may become more difficult with increasing numbers of children. In other words, it is conceivable that, for different reasons, parents who fall somewhere between nulliparity and high parity make optimal health behavior choices; merely having children highlights a need to behave more healthy (Umberson 1987). Whereas, having many children, as Alter, Dribe, and Van Poppel (2007) suggest, may reduce a parent's ability to focus on their own versus their children's needs and subsequently their health and longevity may suffer.

In addition to behavior changes associated with the transition to parenthood, the degree to which parents are socially connected or embedded in social networks which provide access to social support is another important factor associated with parental longevity and mortality (Alter et al. 2007). In particular, aging parents may benefit greatly from the support of multiple children which subsequently helps to improve their own health and reduce their risk of mortality. In addition, nulliparous men may suffer worse health over time compared to parous men, although fathers who live apart from their children may be at a greater risk of mortality

compared to those who co-reside with their children. Using data from the Swedish registry, Weitoft et al. (2004) find that even once health and SES selection processes are controlled, lone men and fathers remain at an increased risk of all-cause mortality and mortality from specific causes.

The Current Study

In the current study, we examine whether the finding that the association between parity and all-cause parental mortality forms a nonlinear association or a J-shaped curve; an association that is neither strictly negative nor strictly positive. In other words, we examine whether the finding holds true (across multiple studies) that low and high parity tend to be associated with a higher risk of all-cause mortality while moderate parity tends to be associated with a lower risk of all-cause parental mortality (Dior et al. 2013; Doblhammer, 2000; Green et al. 1988; Högberg and Wall, 1986; Jaffe et al. 2009). On the one hand, parents' long-term health trajectories may be negatively associated with the number of children they bear and rear, given that more versus fewer children requires greater expenditures of time, energy, and money, net of the time and money that parents spend on themselves. Indeed, as biomedical perspectives suggest, parenthood—especially with increased parity—may increase stress, which is negatively associated with physical and mental health (for a review, see Thoits 2010). On the other hand, parenthood is also associated with benefits and rewards including access to social support from friends and neighbors (Ishii-Kuntz and Seccombe 1989); thus nulliparous (or low parous) adults may have less access to this support and consequently more difficulty dealing with the process of aging.

Indeed, the J-curve hypothesis derives from the interplay of these two competing sets of factors. First, adult children likely provide important social and material support to their

parent(s), which buffers the process of aging (Stein et al. 1998) and which we might expect to increase monotonically as parity increases. Nulliparous adults should not receive this support given that they have no offspring to provide support. Even so, nulliparous adults may receive alternative sources of informal support; however, we might expect that parous adults with large families have at least one child who lives nearby and can provide emotional and instrumental support in times of need. Indeed, Stein et al. (1998) find that adult children, particularly when they are younger, feel a since of obligation to provide support to their parents. Second, parents' economic resources and emotional and physical energies conceivably decline monotonically as parity increases. All else being equal, people who have zero or few children may expend less time, money, and emotional resources outside of expenditures on themselves. Increased parity may therefore result in parental 'depletion', as a result of financial, emotional, and time investments in many children (e.g., Alter, Dribe, and Van Poppel 2007).

Given the interplay between access to social support and physical and economic expenditures on children, we might expect an increased risk of mortality among low and high parous adults, but not for adults with a moderate number of children. In very small families, the lack of support (or a lower amount of support) may offset the fact that parents do not have to share their resources with a large number of children. In very large families, an increased potential for support from children may be offset by an increased likelihood of accumulated economic, physical, and (potentially) emotional depletion as prior research suggests (Alter, Drive, and Van Poppel 2007). People with a moderate number of children may be best situated, in terms of long-term health benefits, because personal resources may be less likely to be depleted, and these parents may also receive adequate support from their children. All else equal, this combination of resources and social and emotional support may ease the aging process, and

potentially reduce the risk of mortality. From a sociological life course perspective, it would follow that parity may indeed affect the overall well-being of parents in differing ways relative to their stage of life (Elder, 1998).

Parents' Gender

While biological and biosocial factors can be intuitively linked with maternal mortality because women bear children and remain the primary caretakers of children (see Casper and Bianchi 2002), the link between parity and mortality is less intuitive for fathers. Research suggests that mothers are more likely to cultivate and reap the benefits of social support associated with longevity (e.g., Barefoot et al. 2005). Yet, other scholars argue that the psychosocial and emotional health of fathers is indeed linked to parity. For example, Weitoft, Burström, and Rosén (2004) found that lone fathers without custody of their children were at an increased risk of mortality. Childless single men were more likely to die early as a result of accidents, suicide, and other forms of violence and were generally more likely to be both addicts and violent.

The literature generally suggests that nulliparity elevates the risk of mortality for single men, while high parity increases the risk of mortality for women for both biological and social reasons. Still, others have found that the nonlinear, or J-curve association between parity and mortality holds for both men and women (e.g., Jaffe et al. 2009). On the other hand, Keizer, Dykstra, and van Lenthe (2011) find that socioeconomic status attenuates the association between parity and mortality among men. Moreover, Penn and Smith (2007) find that women pay a higher cost for fertility compared to men, especially as women age. Overall, based on prior research, we expect to find that the nonlinear association between parity and mortality will be stronger for women than for men.

Social Selection

Social selection may also account, at least in part, for the relationship between parity and all-cause parental mortality (Hurt et al. 2006; Alter et al. 2007). Again, it may be that parous adults are a select group who ultimately change their health-related behaviors upon entering into parenthood, or who are a priori healthier than nulliparous adults. As we noted, research shows that parents versus nonparents are less likely to abuse drugs and alcohol (e.g., Umberson 1987). Even so, it is possible that adults who would otherwise abuse drugs and alcohol refrain from doing so as a direct result of parenthood; and thus parenthood averts unhealthy behaviors and reduces the risk of morality. On the other hand, low parous adults may find time to engage in unhealthy behaviors as fewer children require less expenditure of time and money. It is also conceivable that high parous adults may engage in unhealthy health behaviors to alleviate some of the stress associated with rearing many children.

As we have noted, socioeconomic status may indeed be a source of social selection bias. In addition to less access to nutrition and stress-reducing resources, lower versus higher SES groups are more likely to have high parity and more likely to be at an increased risk of mortality even as they age (Hoffman 2005; Musick et al. 2009). On the other hand, SES does not account for an increased risk of mortality among nulliparous and low parous adults given that higher SES groups are more likely to opt out of childbearing, have fewer children, and live longer than lower SES groups (Casper and Bianchi 2002; Hoffman 2005; Musick et al. 2009). In addition, with respect the nonlinear or J-curve association between parity and parental mortality (both all-cause and cause specific), some studies attempt to account for selection effects, including prior health and measures of SES (e.g., Green, Beral, and Moser 1988; Hurt et al. 2006; Jaffe et al. 2009) and

suggest that, overall, selection accounts for some, but not all, of the association between parity and parental mortality.

Methods

Analytic Approach

To examine the relationship between parity and all-cause parental mortality, we examined data (that we gathered) on 165 independent measures of relative mortality risks (from 30 studies; see Table 1). A meta-analysis model was used to estimate the mean hazard ratio, stratified by the number of covariates. A meta-regression model (a type of weighted linear regression) was used to estimate the effect of covariates on the magnitude of the hazard ratios across sample studies. We assessed the presence and magnitude of heterogeneity using *Q*-tests. All of our analyses were calculated by maximum likelihood using a random effects model (fixed slope, random intercept) and matrix macros provided by Lipsey and Wilson (2001). The possibility of selection and publication bias was examined using a funnel plot, with plot asymmetry evaluated using Egger's test (Egger and Davey-Smith 1998).

[Table 1 about here]

Data Collection and Study Inclusion Criteria

The candidate pool of studies was gathered using an iterative search strategy (Roelfs et al. 2013), beginning with a keyword search in the Medline, EMBASE, CINAHL, and Web of Science databases in 2005 (search terms available upon request) and ending in January 2009 when the hand-search of the literature was completed. The search was designed to capture social support and health studies using any of numerous measures of social support (e.g., social contact frequency, social network size, social participation, social tie proximity, or perceived social support) from a variety of sources ranging from close friends and family to voluntary social

organizations and including support received from children. Figure 1 illustrates our full search and exclusion process. In total, we identified 752 studies which required further examination. Of these, 415 were excluded because all-cause mortality was not the outcome, did not use a relative risk measure, or did not include variables for any of the target measures of social support. The full database of relative mortality risk measures for social support contained information from 337 studies. Of these 337 studies, 270 were excluded because they contained no measure of social support from children and 20 were excluded because they did not specifically measure number of children, but rather only looked at the effects of having versus not having children. Of the remaining publications, 4 were excluded because these studies were conducted in an incomparable, developing nation (i.e., Bangladesh), 4 were excluded because they contained redundant data, and 9 were excluded because they measured mortality during an incomparable time period (prior to 1945). At the end of this process, we were left with 30 studies on which this study is based.

[Figure 1 about here]

Variables

The dependent variable used in the meta-regression (and examined in the meta-analysis) was the log of the relative mortality hazard (i.e., a hazard ratio; the numerator group was respondents with fewer children and the denominator, or comparison group, included respondents with more children). Statistical methods varied between studies, and all non-hazard-ratio point estimates were converted to hazard ratios. Where not reported, standard errors were calculated using (1) confidence intervals, (2) *t* statistics, (3) χ^2 statistics, or (4) *p*-values. We sought to maximize the number of hazard ratios that were analyzed, capturing variability both between and within studies. The focal independent variable was the mean number of children

among the denominator (comparison) group. The number of children was measured in the 30 studies in our analysis using either (in 21 of the studies) discrete categories (e.g., 0-2 children vs. 3 or more children) or (in 7 of the studies) continuous measures (i.e., a count of the number of children). The two additional studies included in the analysis used both discrete and continuous measures. Both types of measures were used for the meta-analyses (both together and separately), but only the discrete measures were used in the meta-regression. This exclusion is based on the observation that the continuous measures provide information about a linear association alone while the central goal of the present paper is to test for a non-linear association.

Where the number of children was measured using discrete categories, we recorded information on the lower and upper boundaries of the categories (see again Table 1) and noted which category was used in the denominator (comparison) group. Assuming a Poisson distribution, we used the information on these lower and upper boundaries to estimate the mean number of children in each discrete category (e.g., a category with a range from 4 to 5 has an estimated mean of 4.38). In cases where the upper boundary of the category was not reported, we conservatively assumed the maximum to be 25 children. The mean number of children for the comparison group and the squared value of the same variable were entered into the regression models in order to examine whether there is a non-linear relationship between number of children and all-cause mortality risk (as some prior research suggests). Descriptive statistics for all variables are reported in Table 2**.**

[Table 2 about here]

The control variables included in the analysis were 1) the difference between the mean number of children for the numerator and denominator groups, 2) the proportion of the sample that was male; 3) the mean age of the study sample, divided by ten; 4) an indicator variable for

whether or not the study sample suffered from a known chronic condition; 5) the underlying death rate in the sample; 6) the duration of follow-up particular to the study; 7) a series of indicator variables for whether or not the study controlled in any way for age, other demographic factors, socioeconomic status, general health status, health-related behaviors (e.g., smoking, drinking), or the presence of chronic health conditions at the individual level; 8) an indicator variable for whether or not the weighting variable for the regression needed to be estimated prior to analysis; 9) a subjective quality rating assigned by the data coders; and 10) a quality rating based on the journal in which a study was published and the relative frequency with which a study had been cited by others.

The difference between the mean number of children for the numerator and denominator groups was included in order to account for how, in some cases, the two groups being compared are adjacent categories (0-1 vs. 2-3 children), while in other cases the two groups are separated more widely (0-1 vs. 6+ children). Sex (measured as the proportion of the sample that was male) was included in order to control for known sex differences in the magnitude of the social support-mortality association. Age (measured as the mean age of the study sample) was included to control for differences in the relative mortality risk due simply to the presence of higher death rates at older ages (ratio comparisons among older samples tend to be closer to 1 because the death rates for both the numerator and denominator groups were high). The indicator variable measuring the presence of a chronic health condition across the entire sample was included because, like the age variable, ratio comparisons among non-healthy samples tend to be closer to 1 because the death rates for both the numerator and denominator groups were high.

We controlled for the underlying death rate for the sample in order to account for any factors other than age or chronic illness that might also affect the magnitude of the relative

mortality hazard in similar ways (i.e., the statistical artifact of being less able to detect differences in hazard rates when death rates are high). Data on death rates was obtained from the *Human Mortality Database* (University of California-Berkeley and Max Planck Institute for Demographic Research 2011). The underlying death rate was then calculated using a weighted average, such that the result would be matched to a particular study in terms of the nation from which the sample was drawn, the year in which the study was conducted, and the gender and age of the respondents.

We controlled for the mean follow-up duration of a study in order to account for differences in the length of time over which mortality could occur. We also controlled for differences in the types of control variables used in each of the articles in our sample by including a series of indicator variables. These are particularly important as we did not use the presence or absence of certain control variables as a factor when making the inclusion/exclusion decisions.

We also included an indicator variable to identify the minority of cases where we had to estimate the weight used for a particular hazard ratio rather than calculate the weight directly from the variance of the hazard ratio (necessary for 15.2% of the hazard ratios included in the analysis). In these cases, the regression weight was estimated using multiple regression from all 337 studies (2,911 hazard ratios) in our social support database. Significant predictors of the standard error were sample size (log transformed), follow-up duration, publication date, the geographic region in which the study was conducted, and an indicator for whether the study controlled for age (Multiple R = .663). We also conducted meta-analyses both including and excluding studies for which we estimated the regression weight. Thus, we retained the ability to assess the impact of regression weight estimation on the final results. Sensitivity tests showed

that there were only minor differences in the results when we excluded the 15.2% of the hazard ratios with estimated inverse variance weights from the analysis. We therefore chose to leave these in the reported analyses, to increase statistical power and our ability to identify important sub-group differences.

Finally, two measures of study quality were adopted. First, we assigned a 3-level subjective rating to each publication. Studies were assigned a low quality rating if they contained obvious reporting or methodological errors (e.g., mathematically impossible confidence intervals or referring to the results of a Poisson regression as an odds ratio). Studies were assigned a high quality rating if the models were well-specified and results were reported in detail. Second, we used principal components factor analysis to construct a scale quality measure using (a) the 5-year impact factor of the journal (the few journals for which an impact factor could not be found were assigned a conservative impact factor of 1; this was done to avoid over-emphasizing their importance, as these were largely second and third tier journals); and (b) the number of citations received per year since publication.

Results

In Table 3, we report the meta-regression results predicting hazard ratio magnitude using a discrete categorical measure of family size. The full modal includes all covariates and the parsimonious model includes only significant covariates. The results of both models suggest a significant non-linear association between the magnitude of the hazard ratio and the mean number of children in the denominator or the comparison group. Both the main effect (p = .076) and the squared term (p = .002) are significant, and the exponentiated coefficients are both smaller than one. The results confirm our primary research hypothesis that there is a curvilinear

J-shaped relationship between parity and all-cause parental mortality. This relationship is shown in Figure 2, which is calculated based on the parsimonious regression model.

[Table 3 about here]

[Figure 2 about here]

As Figure 2 suggests, respondents with fewer children (the numerator group) have a heightened mortality hazard (relative to the denominator group) when parity is low. For example, the mortality hazard is elevated by approximately 42.0% for respondents with zero children when compared to respondents with one child. The degree to which the mortality hazard is elevated declines subsequently, with the mortality hazards between the two groups becoming statistically equal when the mean number of children in the denominator (comparison) group is about 5. As the mean number of children in the denominator group becomes greater than 5, the mortality hazard for the denominator group gradually exceeds the mortality hazard for the numerator group. For example, the hazard rate was 27.9% higher for the denominator group when the mean number of children for this group was seven and the mean number of children for the numerator group was less than seven.

We found no statistically significant difference between men and women for either the linear or non-linear association between mortality and family size. The proportion of the sample that was male did not affect the magnitude of the mortality hazard ($p = .075$), nor did the interaction between sex and the mean number of children ($p = .151$ for the interaction with the main effect; $p = .207$ for the interaction with the squared term). As we discuss below, this suggests the non-linear association between mortality is not accounted for by sex differences (social, biological, or otherwise).

The statistically significant control variables in the parsimonious model included the difference between the mean number of children in the focal (numerator) and comparison (denominator) groups, the underlying death rate for the study, whether or not the study controlled for health behaviors and chronic health conditions, and both measures of study quality. Non-significant covariates included the mean age of the sample ($p = .407$), whether or not an entire study's sample had a chronic health condition of some kind ($p = .618$), the duration of study follow-up ($p = .951$), whether or not the study controlled for age ($p = .119$), other demographic factors ($p = .696$), socioeconomic status ($p = .603$), and general health status ($p = .715$), and whether the inverse variance weight was estimated for a particular hazard ratio ($p = .728$).

In terms of family size, each one unit increase in the difference between numerator group and denominator group (i.e., the difference between the mean number of children for each group) was associated with a 10.2% increase in the magnitude of the hazard ratio ($p < .001$). Including controls for health behaviors such as smoking and drinking was associated with larger hazard ratios (25.1% increase; $p < .001$) while controlling for chronic health conditions was associated with smaller hazard ratios (20.6% decrease; $p < .001$). Finally, the magnitude of the hazard ratios tended to be larger for those studies that we classified as higher quality. Each 1 unit increase in the subjective, coder-assigned quality ranking was associated with a 19.0% increase in the hazard ratio ($p < .001$), while each one unit increase in quality on the citation-based scale was associated with a 4.6% increase in the magnitude of the hazard ratio ($p = .002$).

Table 4 shows a series of mean hazard ratios from our meta-analyses. When the 165 hazard ratios were stratified solely by level of statistical adjustment, among multivariate-adjusted studies, we found the mortality hazard was, on average, 9.2% higher ($p < .001$) for respondents with fewer children when compared to those with more children. Not surprisingly, the difference

in mortality hazard was greater, in relative terms, among studies that only controlled for age (22.4% elevated risk; $p < .001$) or utilized no control variables (24.3% elevated hazard; $p < .001$). There were no significant differences between the subset of hazard ratios based on discrete measures of parity and the subset based on continuous measures.

Table 4 about here

Discussion

We use data from 30 studies and 165 independent measures of relative mortality risks to examine the relationship between parity and all-cause parental mortality and specifically the existence of a J-curve association. Meta-analysis was used to estimate the mean hazard ratio, stratified by the number of covariates. Meta-regression techniques were used to estimate the effect of covariates on the magnitude of the hazard ratios across sample studies. We find that net of covariates, including age, sex, chronic health conditions, and study quality, the mean number of children is associated with all-cause parental mortality. More specifically, our results suggest that there is a significant non-linear association between parity and all-cause parental mortality. That is, low and high parous adults are both at a greater risk of mortality while moderately parous adults appear to be at an advantage in terms of longevity. We further find that the non-linear, or J-shaped, association between parity and all-cause parental mortality is not moderated by parents' gender.

Nulliparous and low parous adults tend to be among higher SES groups (Casper and Bianchi 2002), and therefore have financial resources which are useful as adults age. Even so, our findings suggest that the long-term consequences of nulliparity or low parity may be that the social connections between parents and children over their life course are important beyond access to financial resources. Intuitively, adequate levels of emotional and/or instrumental social

support may not be the type of resources that can be bought. Yet, research suggests that aging populations benefit from receipt of social support (e.g., Lyyra & Heikkinen 2006; Avlund, Damsgaard, and Holstein 1998) and access to support increases parents' ability cope with the onset of diseases and/or disabilities associated with aging (e.g., Penninx et al. 1997). Research further suggests that adult children provide a substantial proportion of social support to their aging parents (e.g., Stein et al. 1998). Moreover, research further suggests that social isolation (i.e., little to no access to support) increases the risk of mortality (see House, Landis, and Umberson 1988; House 2001). To the extent that nulliparity and low parity increase the risk of social isolation, particularly following the loss of a spouse or for those who never marry, it may be that parity operates through social isolation to influence adults' risk of mortality (e.g., see Roelfs et al. 2011; Shor et al. 2012). While our data do not allow us to test directly this hypothesis, future studies may consider the potential mediating influence of social isolation in the association between parity (particularly nulliparity and low parity) and parental mortality.

On the other end of the parity distribution, research suggests that low-income adults are more likely to have more children (Casper and Bianchi 2002) and also more likely to be at an increased risk of mortality (Hoffman 2005; Musick et al. 2009). Given these associations, it is difficult to rule out the possibility that the influence of parity on the risk of mortality (found in this study and prior studies) is accounted for primarily by selection. We attempt to address this possibility and, consistent with what others have found (e.g., Jaffe et al. 2009), our results suggest that there is a nonlinear association wherein those with high parity are at an increased risk of mortality net of selection. With respect to social support, intuitively, we would expect high parity to be associated with greater access to social support as parents would have more children from whom to receive help as they age. On the other hand, high parity increases stress-

related health problems or other chronic illness (Alter, Dribe, and Van Poppel 2007) which increase the risk of mortality. In addition to which, low-income parents are more likely to bear and rear children across partnerships (so-called multipartnered fertility), which Harknett and Knab (2007) found to be associated with less access to perceived social support.

Overall, consistent with prior research (Dior et al. 2013; Doblhammer 2000; Green et al. 1988; Högberg and Wall 1986; Jaffe et al. 2009), we find that low and high parity increase significantly the risk of all-cause parental mortality. Moreover, based on prior research which suggests that pregnancy influences women's postpartum health and risk of infection/disease, and that parity differentially affects the longevity of men and women (Alter, Dribe, and Van Poppel 2007; Daling 2002; Dribe 2007; Henretta 2007; Penn and Smith 2007), we examine whether the J-curve association between parity and all-cause parental mortality is moderated by gender. Both the non-significance of the main effects and the interactions between sex and parity suggest that the shape, direction, and significance of the non-linear association between parity and all-cause mortality are similar for men and women. This finding is inconsistent with prior research which suggests that parity influences women's versus men's longevity more strongly (e.g., Penn and Smith 2007).

It may be that the social factors associated with all-cause parental mortality vary less by gender compared to the physical factors associated with mortality. That is, while men versus women may be at an increased risk of chronic disease onset (for discussion, see Bird and Rieker 1999), parity and relationships with offspring (over time) may similarly situate aging parents with respect to access to social support. That is, aging mothers and fathers may receive (or not receive) similar levels and types of social support from their adult children. Along these lines, Bird and Rieker (1999) convincingly argue that to better understand the differences in health

outcomes among women and men, scholars must account for *both* biological and social influences. Future research may shed light on the extent to which physical well-being, long-term relationships with adult children, and all-cause parental mortality are linked.

Limitations

As is the case with most studies, our study is not without limitations. Caution should be taken when interpreting the mean hazard ratios reported in our results given the presence of data heterogeneity and the possibility of at least some publication/selection bias. The null hypothesis of data homogeneity was rejected (at the .05 level) for five of the eleven mean hazard ratios. This suggests that important between-study differences exist; a major reason for focusing on the results of the meta-regression rather than the meta-analysis. The results of the Egger's test for funnel plot asymmetry (p = .065; see Figure 3) indicated a marginally acceptable level of publication/selection bias in the data. A visual examination of the funnel plot suggests the missing studies had small sample sizes and would have reported a relative hazard rate for respondents with fewer children that may have been more elevated than the average found in our analysis. Thus, the meta-analysis and meta-regression results can be viewed as conservative estimates of the relative mortality hazard associated with having fewer children.

Overall, this study suggests that the long-term consequences of parity are more strongly associated with the social connections between parents and their children rather than the biological or biomedical consequences. Our findings show that parents who have a moderate number of children (versus too few or too many) live longer; and while we are unable to test directly the reasons why, we speculate that moderately parous adults may benefit from expending a manageable amount of resources on their children, experiencing a manageable level of stress associated with childrearing, and receipt of adequate levels of social support from their

children as they age. The implications of this study are that early life choices associated with childbearing have long-term consequences for parents, particularly given that children's and parents' lives are linked across each of their life courses (Elder 1998).

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Table 1. Summary information for studies included in the analysis					
Authors	*Country*	*Study Years*	*Measurement Used*	*Number of HRs Used*	*Sample Size*
Daling et al. 2002	United States	1983-2000	Discrete Categories (0, 1, 2-3, 4+)	3	1,174
Dior et al. 2013	Israel	1964-2005	Discrete Categories (1, 2-4, 5-9, 10+)	3	40,454
Doblhammer 2000	United Kingdom	1971-1996	Discrete Categories (0, 1-2, 3+)	2	56,164
	Austria	1981-1997	Discrete Categories (0, 1, 2, 3, 4, 5+)	5	1,254,153
Green et al. 1988	United Kingdom	1971-1981	Discrete Categories (0, 1, 2, 3, 4, 5+)	5	108,352
Grundy and Kravdal 2008	Norway	1980-2003	Discrete Categories (0, 1, 2, 3, 4, 5+)	20	1,530,101
Guilley et al. 2005	Switzerland	1994-1999	Discrete Categories (0, 1+)	1	295
Henretta 2007	United States	1994-2002	Discrete Categories (0, 1, 2, 3, 4, 5+)	5	4,335
Hermalin et al. 2009	Taiwan	1989-2003	Continuous Count	2	4,049
Jaffe et al. 2009	Israel	1995-2004	Discrete Categories (0, 1, 2, 3-4, 5-7, 8+)	20	134,555
Jylha and Aro 1989	Finland	1979-1985	Discrete Categories (0, 1+)	4	1,060
Koski-Rahikkala et al. 2006	Finland	1965-2001	Discrete Categories (1, 2-4, 5-9, 10+)	3	12,055
Kotler and Wingard 1989	United States	1965-1982	Discrete Categories (0, 1-3, 4+)	6	3,188
Kravdal 2003	Norway	1960-1999	Discrete Categories (0, 1-2, 3+)	4	3,638
Kroenke et al. 2006	United States	1992-2004	Discrete Categories (0, 1-2, 3-5, 6+)	3	2,835
Kvale et al. 1994	Norway	1961-1980	Discrete Categories (0, 1, 2, 3, 4, 5+)	10	63,090
	Norway	1961-1980	Continuous Count	2	
Lund et al. 1990	Norway	1970-1985	Discrete Categories (0, 1, 2, 3, 4, 5, 6+)	29	822,593
	Norway	1970-1985	Continuous Count	6	
Manor et al. 2000	Israel	1983-1992	Discrete Categories (0, 1, 2, 3, 4-5, 6+)	10	79,623
Martikainen 1995	Finland	1980-1985	Continuous Count	1	4,779,535
Menotti et al. 2006	Italy	1960-2000	Discrete Categories (0, 1+)	1	1,712
Mohle-Boetani et al. 1988	United States	1973-1985	Discrete Categories (0, 1+)	1	838
Olson et al. 1998	United States	1978-1992	Discrete Categories (0, 1-2, 3+)	2	540
Smith and Zick 1994	United States	1968-1987	Continuous Count	2	2,604
Spence 2006	United States	1967-2001	Continuous Count	1	3,258
Sun and Liu 2008	China	1998-2000	Continuous Count	1	7,938
Trivers et al. 2007	United States	1990-2000	Discrete Categories (0, 1-3, 4+)	2	1,264
Villingshoj et al. 2006	Denmark	1991-2002	Continuous Count	1	770
Walter-Ginzburg et al. 2002	Israel	1989-1997	Continuous Count	1	1,340
Weitoft et al. 2000	Sweden	1990-1995	Discrete Categories (1, 2-3, 4+)	4	712,479

Weitoft et al. 2004	Sweden	1990-2000	Continuous Count	1	682,919
Yasuda et al. 1997	United States	1984-1994	Discrete Categories (0, 1-2, 3+)	4	806

Table 2. Descriptive Statistics [1]			
	Minimum	*Maximum*	*Mean / %*
Mean number of children in comparison group [2]	1	10.35	3.67
Difference between mean number of children in the focal and comparison groups [2]	1	7.44	3.00
Gender of the sample			
Male only			18.2%
Female only			79.4%
Mixed gender			2.4%
Mean age of the sample	28	92	54.99
Chronic health condition for sample (1=yes)			1.2%
Underlying death rate (per 1000 population)	0.56	837.60	78.97
Follow-up duration (years)	1	40	14.65
Study controlled for:			
Age			77.0%
Other demographic factors			35.2%
Socioeconomic status			55.8%
General health status			17.6%
Health behaviors (smoking, drinking, etc.)			9.7%
Chronic conditions			12.7%
Regression weight estimated (1=yes)			15.2%
Study Quality:			
Coder rating (3-level scale)	Average (2)	High (3)	2.85
Citation-based scale	0.22	5.27	1.32

[1] N = 165 hazard ratios (147 using a categorical measure and 18 using a continuous measure of number of children), except where otherwise indicated
[2] Based on N = 147 hazard ratios using a categorical measure of number of children

Table 3. Meta-regression models predicting HR magnitude among studies of number of children vs. all-cause mortality (lower number vs. higher number) using a discrete categorical measure of family size [1]

	Full Model	*Parsimonious Model*
Constant	0.650 (.085)	0.662 (.005)
Mean number of children in comparison group	0.976 (.369)	0.960 (.076)
Mean number of children in comparison group, squared	0.992 (.002)	0.993 (.002)
Proportion of the sample that was male (sex)	1.240 (.075)	…
Interactions		
Sex * Mean number of children in comparison group	0.918 (.151)	…
Sex * Mean number of children in comparison group, squared	1.008 (.207)	…
Difference between mean number of children in the focal and comparison groups	1.093 (<.001)	1.102 (<.001)
Mean age of the sample (divided by 10)	1.011 (.407)	…
Chronic health condition for sample (1=yes)	1.076 (.618)	…
Underlying death rate	1.000 (.012)	1.000 (.004)
Follow-up duration (years)	1.000 (.951)	…
Study controlled for:		
Age	1.068 (.119)	…
Other demographic factors	0.983 (.696)	…
Socioeconomic status	0.980 (.603)	…
General health status	0.977 (.715)	…
Health behaviors (smoking, drinking, etc.)	1.349 (.012)	1.251 (.004)
Chronic conditions	0.810 (.009)	0.794 (.002)
Regression weight estimated (1=yes)	0.981 (.728)	…
Study Quality:		
Coder rating	1.143 (.047)	1.190 (<.001)
Citation-based scale	1.058 (.007)	1.046 (.002)

[1] Numbers reported are exponentiated regression coefficients (p-value in parentheses). Exponentiated regression coefficients represent a ratio of a HR at one level on the IV to the HR at the next lowest level. Ellipses indicate when a variable was not entered into the model. N = 147 hazard ratios. R^2 = .46 for the full model and R^2 = .43 for the parsimonious model.

Table 4. Meta-analyses of the association between number of children and all-cause mortality									
	Unadjusted HRs			*Age-adjusted HRs*			*Multivariate-adjusted HRs* [1]		
	Mean HR	*Number of HRs*	*Q-test p-value* [2]	*Mean HR*	*Number of HRs*	*Q-test p-value* [2]	*Mean HR*	*Number of HRs*	*Q-test p-value* [2]
All HRs	1.243***	24	.0446	1.224***	37	.6739	1.092***	104	.0779
Excluding HRs where weight was estimated	1.286***	14	.0114	1.223***	37	.7529	1.094***	89	.0403
Discrete measures only	1.272***	16	.0433	1.224***	37	.6271	1.090***	94	.0419
Continuous measures only	1.214**	8	.2576	…	0	…	1.106	10	.5603

[1] Covariates vary between studies
[2] Q-test p-value refers to Cochrane's Q, a measure of heterogeneity among the effect sizes within a group.
* p-value < .05 ** p-value < .01 *** p-value < .001

Figure 1. Study inclusion/exclusion flow diagram

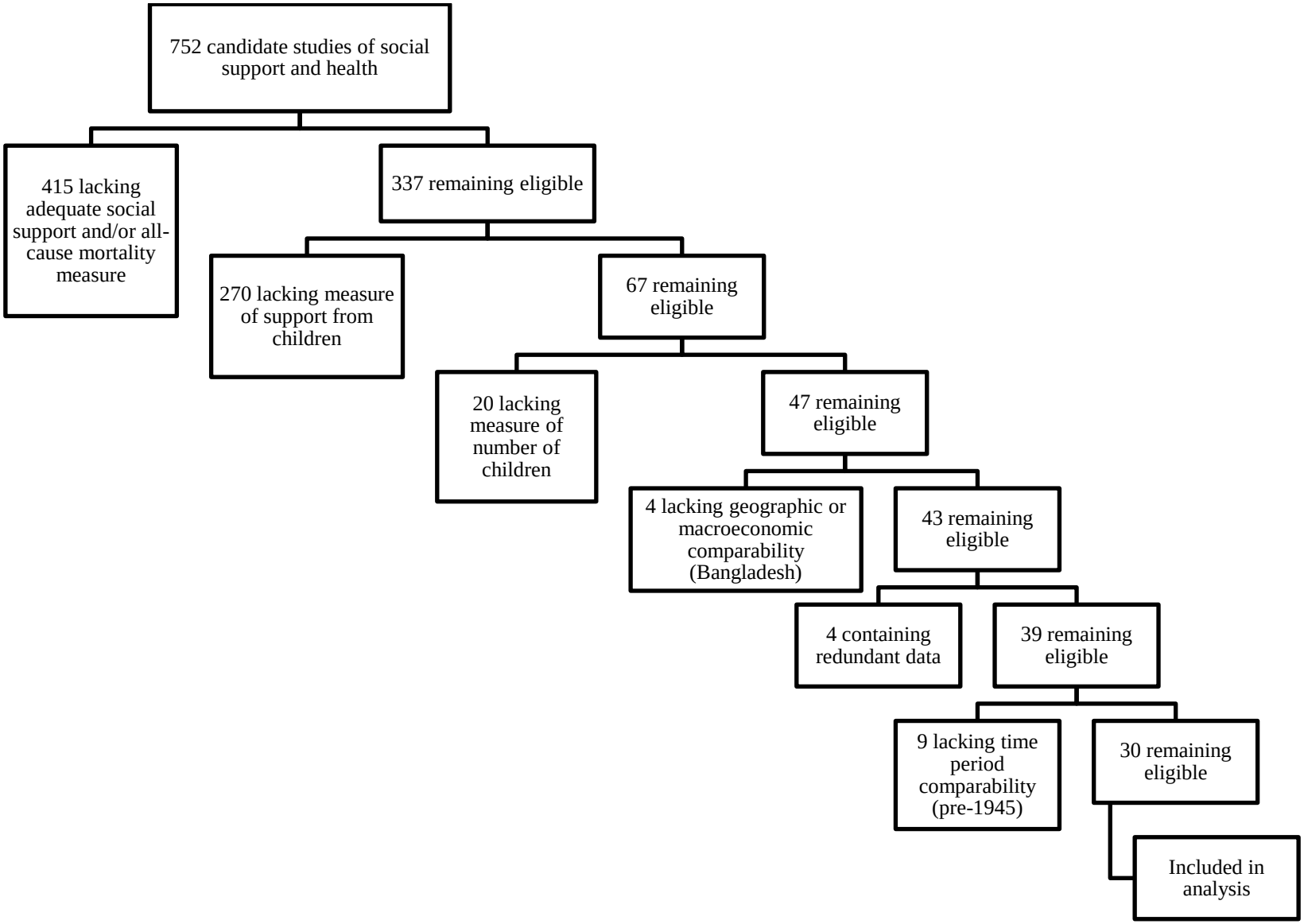
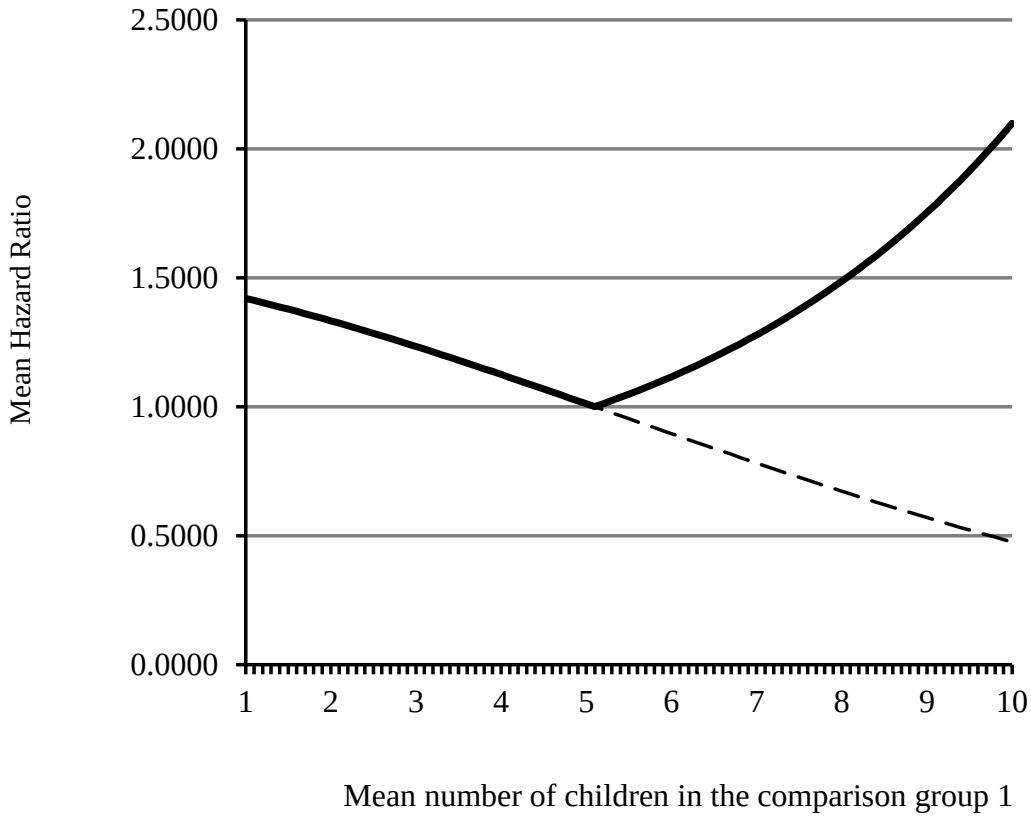
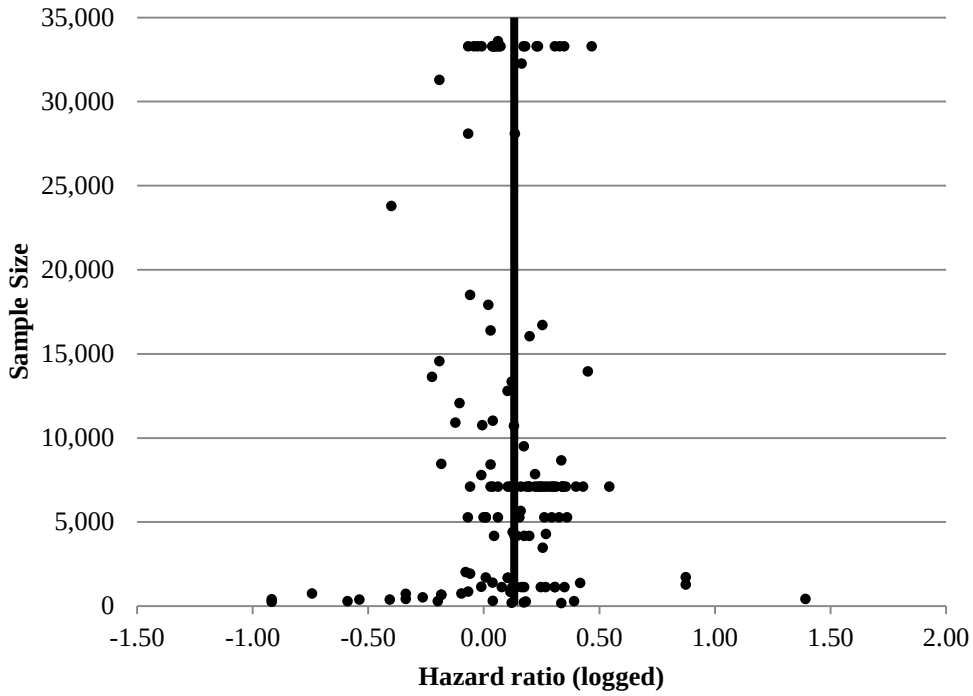


Figure 2. Mean hazard ratio by the mean number of children in the comparison group



[1] For each pairwise comparison, the comparison group consists of those with a greater number of children. The solid line represents the mean hazard ratio when the group with the highest risk of mortality (case or comparison group) is always placed in the numerator (comparison group in the denominator up to a mean number of children = 5.0 and in the numerator thereafter). The dashed line represents the mean hazard ratio when the comparison group is always placed in the denominator.

Figure 3. Funnel plot of hazard ratios (logged) vs. sample size [1]



[1] Vertical line denotes the mean hazard ratio (logged) of 0.1321 among the 147 hazard ratios from studies using a categorical measure for number of children; plot excludes 9 HRs with sample sizes of 90,000 or greater in order to show detail of the variability at smaller sample sizes. P-value from Egger's test for funnel plot assymetry = .011.