

Extrinsic Mortality Effects on Reproductive Strategies in a Caribbean Community

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Published online: 16 June 2010
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Abstract Extrinsic mortality is a key influence on organisms' life history strategies, especially on age at maturity. This historical longitudinal study of 125 women in rural Domenica examines effects of extrinsic mortality on human age at maturity and pace of reproduction. Extrinsic mortality is indicated by local population infant mortality rates during infancy and at maturity between the years 1925 and 2000. Extrinsic mortality shows effects on age at first birth and pace of reproduction among these women. Parish death records show huge historical variation in age-specific mortality rates. The infant mortality rate (IMR) in the early 1920s was low, increased dramatically beginning in 1929, and reached a maximum in the 1950s, at which point IMR declined steadily to its present low rate. The mortality rate early in life showed a quadratic association with age at first birth. Women who experienced conditions of low IMR early in life reproduced relatively late in life. Those born into moderately high levels of infant mortality tended to reproduce earlier than those born at low levels. At very high infant mortality levels early in life, women went on to delay reproduction until relatively late, possibly as a result of somatic depletion and energetic stress associated with the conditions that lead to high IMR. Population mortality rates at age of maturity also showed a quadratic association with age at first birth. The pace of reproduction, estimated as number of surviving offspring controlled for maternal age, showed a similar quadratic effect. There were complex interactions between population mortality rates in infancy and at maturity. When extrinsic mortality was high during infancy, extrinsic mortality later in life had little effect on timing of first birth. When extrinsic mortality was low to moderate in infancy, extrinsic mortality later in life had significant effects on adult reproduction. I speculate that these effects are mediated through development of personality facets associated with reproduction.

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Keywords Risk · Teen pregnancy · Child development · Evolutionary ecology · Behavioral ecology · Demography · Personality

Extrinsic mortality is the risk of death that is not conditional on an organism's reproductive behavior (Stearns 1992:182). We can define extrinsic mortality as variance in the probability of death that is not accounted for by mating or parenting effort (or, by extension, trade-offs between reproductive and somatic effort). In other words, an organism cannot escape extrinsic mortality by changing its behavior: it is the age-specific risk of death that is equally shared by all members of a population. Intrinsic mortality, in contrast is the probability of death associated with allocation of somatic and reproductive effort. Predation, for example, could result in either extrinsic or intrinsic mortality, or both. Imagine a population of organisms in which there is a probability (p) of death from predation at age x . Then p is a combination of factors, some of which are beyond an individual's control but others are not. The frequency by which an individual encounters a predator depends on extrinsic factors, such as the density of predators in the environment (beyond the individual's control), and intrinsic factors, such as the level of vigilance, time spent exposed in the landscape as a result of mating effort, and so on (determined by allocation of effort). An individual of a prey species in an environment with many predators may reduce the probability of death by predation by adjusting its behavior, but there is always some extrinsic probability of death by predation. The predation example raises an important point about extrinsic mortality: Any age-specific probability of death has both intrinsic and extrinsic components that can be difficult to isolate analytically. Despite empirical challenges, extrinsic and intrinsic components of mortality can have profound influences on adaptive behavior.

Extrinsic mortality plays a key role in the evolution of life histories and reproductive strategies (Chisholm 1993, 1999a; Promislow and Harvey 1991; Roff 2002; Stearns 1992). When extrinsic mortality is high, organisms should reproduce early in life to reduce mortality exposure per unit of time and extend the length of the reproductive span, which should maximize fertility to "beat the odds" that some offspring will die. Conversely when extrinsic mortality is low, then differential reproductive success is contingent on resources invested in growth, development, and parental effort rather than luck. Hence, in low extrinsic-risk environments, individuals may enhance fitness by delaying reproduction to accrue additional resources (including knowledge and skills), and by reducing fertility and increasing investment per offspring. These predicted relationships hold among mammals: Juvenile mortality is negatively correlated with age at maturity, age at weaning, and maternal investment, and positively correlated with litter size and pace of reproduction (Promislow and Harvey 1990:424).

Extrinsic risk for humans has attracted theoretical interest since the early 1990s (e.g., Borgerhoff Mulder 1992; Chisholm 1993, 1999a; Harpending et al. 1990); however, empirical work is relatively scarce. Several studies show predicted relations between extrinsic risk and human life history patterns. Mortality was shown to be negatively associated with age at reproductive maturity among urban Americans (Wilson and Daly 1997), sub-Saharan Africans (Gant et al. 2009), and in two cross-national studies (Low et al. 2008; Walker et al. 2006). Extrinsic risk predicts patterns of

parental care cross-culturally (Quinlan 2006, 2007). And perception of mortality may influence human reproductive behavior (Chisholm et al. 2005). Even though this body of research is small, it seems clear that local extrinsic risk is an important environmental cue for shaping human reproductive strategies, but how and when are local environmental conditions encoded into life histories?

What role do environmental cues play in the development of human reproductive strategies, and when in development are those cues most salient? Long-standing debate in anthropology and evolutionary psychology identifies two important periods for shaping the adult phenotype. Early childhood, from 1 to about 7 years of age, has been suggested as a sensitive period for development that has strong effects on adult outcomes (e.g., Belsky et al. 1991; Chisholm 1999a; Draper and Harpending 1982; Ellis et al. 1999; Quinlan 2003; Quinlan and Quinlan 2007). Recent adoption studies find that conditions in the first 42 months are important in shaping development during childhood, with a dose-response effect for duration and severity of environmental conditions (Rutter and O'Connor 2004). In these related early childhood models, caregiver responsiveness emerges as one likely mechanism that communicates environmental conditions to developing offspring. In harsh and risky environments, parents themselves engage in high-fertility, low-investment reproductive strategies that include relatively unresponsive parental care (Quinlan 2007). Theoretically, this pattern of “low-intensity” parental care switches children toward a similar low-investment, high-fertility developmental trajectory. The psychological and physiological mechanisms are unclear, but psychosocial stress, attachment and associated hormones have been implicated (Chisholm 1999a; Ellis 2004; Flinn et al. 2008; Schechter and Francis 2010). The parental responsiveness—early childhood theory of reproductive development suggests strong vertical or intergenerational transmission of adult phenotypes (Quinlan and Flinn 2003).

A second, though not necessarily mutually exclusive, line of thought suggests that it makes little adaptive sense to lock humans into an adult phenotype early in the life course because such canalization could lead to significant mismatch between adult phenotypes and the extant environment (Quinlan and Flinn 2003; Whiting 1980). Hence, humans should be open to environmental influences throughout the life course, and conditions at the age of maturity should be particularly salient in shaping behavior. Research has shown that stressful family events like parental separation in early childhood (birth to about age 6 years) have the strongest effects on the development of adult phenotypes, and the effect diminishes when those events occur later in adolescence (Quinlan 2003). Stressful family life, however, may become a less salient environmental cue later in development as other information becomes more pertinent. Hence, strong effects later in life (around age at maturity) are predicted in addition to early developmental effects. Mechanisms for later developmental effects are unknown, but physiological pathways (see Vitzthum 2001) and conscious decision making are likely important.

The present study cannot address mechanistic questions; instead it focuses on timing of environmental cues by examining effects of extrinsic mortality on timing of first birth and pace of reproduction for a rural community in the Commonwealth of Dominica. The study takes advantage of a naturalistic experiment (see Rutter 2007) in an extraordinarily variable environment that occurred between 1925 and 2001 to examine population-level extrinsic mortality rates for year of birth and year

at maturity (first birth) for 125 rural Dominican women. When extrinsic mortality is high, age at first birth is predicted to be relatively early and pace of reproduction will be relatively rapid. When extrinsic mortality is low, age at first birth is predicted to be relatively late and pace of reproduction will be relatively slow. These basic predictions are perhaps too simplistic.

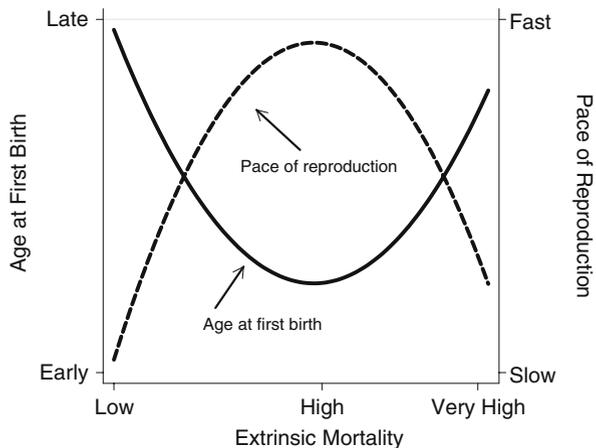
Very high extrinsic mortality rates may indicate an environment that is so harsh that reproduction is not feasible (Ellis 2004). Hence, at very high extrinsic mortality rates, individuals may shut down reproductive development to preserve somatic resources in hopes that the environment may improve later. Extrinsic mortality, hence, is predicted to show quadratic associations with reproductive behavior (Fig. 1).

The Site

The substantial variation in historical infant mortality rates in rural Dominica presents an instructive case study for the effects of extrinsic mortality on reproductive strategies. The Commonwealth of Dominica is a small, rural island nation located between Guadeloupe and Martinique (15°N, 61°W). The island is mountainous and relatively undeveloped. Dominica's population (approximately 65,000) is of mixed African, European, and Island-Carib descent. Most Dominicans are bilingual in English and French-Patois. Dominica is part of the British Commonwealth of Nations and received independence from colonial rule in 1978.

Bwa Mawego, the study site, is one of the least developed villages on the remote, windward side of the island in St. David Parish. Of the approximately 700 full- and part-time residents, about 400 people were in residence at the time of the most recent census (unpublished data collected by the author). Average annual household income in Bwa Mawego is approximately \$5,000 East Caribbean Dollars (US \$1,850). Economic activities include subsistence gardening, fishing, bay oil production, banana production, running a rum shop, and limited wage labor. Most adults are involved in subsistence horticulture. Bay oil, from bay leaf or *bwaden*, is the most

Fig. 1 Predicted relations between intrinsic mortality and reproductive strategies. (Also see Ellis 2004)



important source of cash for most people in Bwa Mawego, but other cash crops, including bananas, coffee, and limes, were important in the past. Today the population is relatively healthy, though conditions were much worse in the recent past. The infant mortality rate (IMR) for the island is 17 per 1,000 live births compared with 46 per 1,000 for the Caribbean region as a whole (U.S. Census Bureau 2008); however, IMR for St. David Parish reached a remarkable and tragic high of 724 per 1,000 in 1950 (discussed below). Opportunities for education are limited. About 30% of villagers born between 1953 and 1986 have attended “high school,” which is approximately equivalent to the ninth and tenth grades in the U.S. Almost no older individuals attended high school. Kinship and family are the foundation of economic, social, and reproductive behavior in Bwa Mawego. Almost everyone in the village is related through blood or marriage. Kin ties provide a map for navigating social life, and they offer avenues for the flow of goods and services. Family members cooperate for construction and agricultural projects. Related women share childcare duties. Unrelated friends are also important, but kin have priority in Bwa Mawego. More detailed descriptions of the site can be found in Quinlan (2004), Quinlan and Flinn (2005), and Quinlan (2006).

Methods

Infant mortality rates (IMR) indicating extrinsic risk were calculated from St. David Parish birth and death records for the years 1925 through 1988 and by censuses and household monitoring from 1988 to 2000. The Catholic Church was officially responsible for maintaining birth and death records in the Commonwealth of Dominica until the mid-1980s, when women were required by law to give birth at Princes Margaret Hospital in the capital city. Parish death records show little evidence of reporting biases: The handwriting is consistent over many years, and changes in handwriting are not associated with substantial changes in IMR, indicating a single recorder of vital events for the parish even during several years showing high variation. Furthermore, changes in IMR are consistent with documented historical events (such as food shortages and, later, public health campaigns, reported below) and ethnographic accounts.

Here IMR is defined as deaths by 1 year of age divided by total births for the year. For 1925 through 1988, IMR was calculated for the entire parish, which includes about eight villages along approximately 7 miles of the rural Atlantic coast. After 1988, IMR was calculated for two villages for which we have complete genealogical and demographic records (collected by M. Flinn and R. Quinlan). IMR is used rather than other measures of mortality because the number of infants at risk for the year is easily and reliably calculated, whereas “at-risk” populations for other age groups are more difficult to document because of migration. Also, because of infants’ vulnerability, IMR is a fairly sensitive assay of environmental conditions. In sum, the IMR measure used here provides a good estimate of juvenile mortality, which has been shown to have strong effects on life history (Charnov 1991; Promislow and Harvey 1990). IMR was calculated for each woman’s year of birth and in the year in which she gave birth for the first time: IMR in year of birth is a measure of

population mortality early in life;¹ IMR in the year the woman gave birth is a measure of population mortality risk at age of maturity.

Age at first birth was also calculated from parish records. Women who had never given birth were excluded from the analysis. Reproductive success (RS) was calculated from genealogies that include all offspring surviving to age 10 years (see Quinlan and Hagen 2008). RS was controlled for maternal age in linear regressions, which indicates the pace of reproduction as births per year per woman. RS was a linear function of age (quadratic terms were not statistically significant, and inclusion of quadratic terms did not appreciably change the shape or slope of the RS–maternal age function).

Results of multivariate analyses are from OLS multiple linear regression and censored Poisson regression. Because the criterion variables are in counts (years to first birth, and number of surviving offspring), Poisson regression is the preferred method; however, comparison of means, medians, ranges, and standard deviations for RS and age at first birth indicate roughly normal distributions (Table 1). Poisson regression for RS and age at first birth gave results with very similar effects to OLS. OLS results are reported alone for age at first birth because that measure closely approximates a normal distribution (Table 1). OLS results are presented here along with Poisson coefficients for RS (see below) because RS has a low mean close to the standard deviation (characteristic of a Poisson distribution); however, OLS coefficients give a more intuitive sense of the outcomes. Otherwise the models conform to the assumptions of OLS regression. Alpha was set at 0.05 for first-order effects, and 0.10 for quadratic and interaction effects to compensate variation inflation common with those higher-order terms.

Extrinsic Mortality in Rural Dominica

Currently the Commonwealth of Dominica has one of the healthiest populations in the Caribbean region, but that was not always the case. St. David Parish death records going back to the early 1920s show huge variation in age-specific mortality rates. Infant mortality in the early 1920s was low, but IMR increased dramatically around the beginning of the global economic depression in 1929 (Fig. 2). The cause of the IMR spike in 1929 is not clear, though a flu epidemic (a regular occurrence in Bwa Mawego) exacerbated by deteriorating economic conditions (Honeychurch 1995:160–61) is one likely explanation. Infant mortality rates were high and variable from 1929 throughout the 1950s (Fig. 2); however, rates were astronomically high between 1943 and 1956 with a peak of 72% in 1950. During this period Dominica experienced food shortages related to World War II shipping disruptions and slow postwar reconstruction. Locals recount an extreme shortage of meat (usually imported) and fish owing to disruptions in normal fishing activities as result of the “Battle of the Caribbean” (Whitham 2002:65–85). The joint U.S. and British Caribbean Commission reported widespread food shortages in the Caribbean and “famine” on Dominica during the war years (Whitham 2002).

¹ Because IMR is calculated for birth year and some children were born at midyear or later, we cannot rule out gestational effects as well as effects during early infancy.

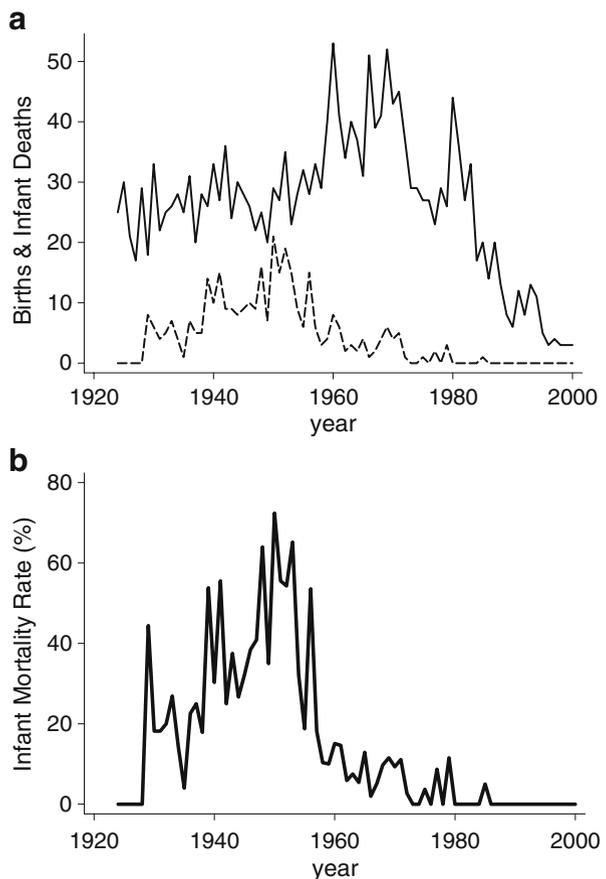
Table 1 Descriptive statistics for criterion and predictor variables used in OLS regression

	N	Mean	Median	SD	Min.	Max.
Year of birth	127	1950.93	1952	14.99	1924	1984
Year of first birth	127	1971.51	1971	14.05	1938	2001
Reproductive success	127	5.33	5	2.88	0	13
Age at first birth	125	20.74	20	3.60	14	31
IMR in year of birth (%)	127	22.45	15.09	18.79	0	72.41
IMR in year of first birth (%)	127	11.53	7.50	16.62	0	65.22

In response to food shortages, villagers in Bwa Mawego turned to raising pigs as a source of protein. Unfortunately, these rural villagers had little experience in pig husbandry; the pig population grew rapidly and was more-or-less free range, with pigs living among people in their yards and roaming freely throughout the village, including the drinking water supply. One informant describes the situation succinctly: “There was pig shit, pig shit, pig shit everywhere.” While I cannot be

Fig. 2 Historical trends in infant mortality in rural Dominica.

(a) births (*solid line*) and infant deaths (*dashed line*). Note that part of the downward trend in births is due to the smaller population observed after 1988 (see [Methods](#)). (b) IMR



sure, it seems possible that the incredibly high IMR in 1950 coincides with the peak of the pig population in Bwa Mawego. Later in the 1950s, development efforts reached the village, including a public health campaign that placed strong legal restrictions on keeping pigs. Apparently those restrictions were taken quite seriously, and by the early 1990s many villagers I knew said it was illegal to keep pigs in the village, and they expressed real concern over the few villagers who still kept pigs.

Effects of Mortality on Life History

Correlations among IMR at birth, IMR at maturity, and year of birth are presented in Table 2. Because of the tremendous variation in IMR over the years examined, there is no association between IMR at birth and at maturity ($r^2 < 0.01$), which provides an interesting natural experiment for effects of extrinsic risk through the life course. IMR in infancy and year of birth showed a modest negative correlation ($r = -0.26$) with substantial “unaccounted for” variance owing to the variability in IMR. IMR at maturity and year of birth showed a more substantial negative correlation ($r = -0.69$), indicating the general downward trend in population IMR after 1950. This latter correlation suggests problematic multicollinearity in the regression models. However, the maximum Variance Inflation Factor (VIF) for models presented in Tables 3 and 5 (after removing quadratic effects, which inevitably result in substantial VIFs) was 2.5, indicating that multicollinearity was not a problem in these models.

Population IMR during mother’s year of birth showed a quadratic association with her age at first birth (Table 3, Fig. 3). Under conditions of low mortality, women reproduced relatively late in life. At moderately high levels of mortality women tended to reproduce earlier than under low levels. At very high mortality levels in early life women went on to delay reproduction until relatively late, possibly as a result of somatic depletion and energetic stress associated with the conditions leading to high IMR (Ellis 2004). Population mortality rates at maturity showed the same quadratic association, although mortality at maturity accounted for more of the variance in age at first birth (19%) than did mortality in infancy (13%, Table 3).

Complex interactions between population mortality rates in infancy and at maturity for the effect on age at first birth are indicated in Fig. 4. The graphic representation in Fig. 4 should be taken as an approximation of the interaction reported in Table 3. The graph was produced from a series of separate regression models for four intervals of IMR at birth. OLS estimates for the means of each interval are given in Table 4. When extrinsic mortality was high in infancy, extrinsic mortality later in life had little effect on adult reproduction. When extrinsic mortality

Table 2 Correlations between IMR in year of birth and IMR in year of first birth

	Year of birth	IMR at birth
Year of birth	1.00	
IMR at birth	-0.26	1.00
IMR at first birth	-0.69	-0.02

Table 3 Multiple linear regression showing effects of IMR on age at first birth

AFB	B	<i>p</i>	r_p^2	R_p^2 quadratic IMR & interaction effects
(Constant)	601.15	0.00		
Year of birth	-0.30	0.00	0.31	
IMR at birth	-0.19	0.00	0.07	
IMR at birth squared	0.002	0.01	0.05	0.13 ^a
IMR at first birth	-0.34	0.00	0.11	
IMR at first birth squared	0.004	0.00	0.07	0.19 ^a
IMR × IMR	0.004	0.01	0.05	0.05 ^b
		$R^2=0.68$		0.37

B = unstandardized regression coefficient; *p* = *p*-value for unstandardized regression coefficient; r_p^2 = partial Pearson’s correlation coefficient squared; the last column shows the coefficient of determination for quadratic effects. IMR × IMR with highest value Winsorized to the next highest value yielded $r_p^2=0.04$; other effects were basically unchanged

^a R_p^2 for IMR and IMR-squared

^b r_p^2 for the interaction effect

was low to moderate in infancy, extrinsic mortality later in life had significant effects on adult reproduction.

The pace of reproduction estimated as number of surviving offspring adjusted for maternal age showed a similar, but inverted, quadratic effect (Table 5, Fig. 5). Reproductive success increased as IMR increased, but decreased at very high mortality levels. No doubt, the strong downward trend in RS at IMR rates above 40% reflects the loss of children as well as (if not rather than) a change in fertility. However, the increase in RS at higher levels of IMR—especially IMR in mother’s infancy—offers strong evidence for changes in reproductive behavior associated with population-level mortality rates: Women can only increase RS in the face of rising infant mortality by increasing fertility. This association suggests a substantial increase in fertility associated with IMR; however, the model in Table 5 indicates that IMR only accounts for 16% of the variance in RS. Again, IMR at maturity was a

Fig. 3 Effects of IMR in infancy and at maturity on age at first birth ($R^2=0.32$)

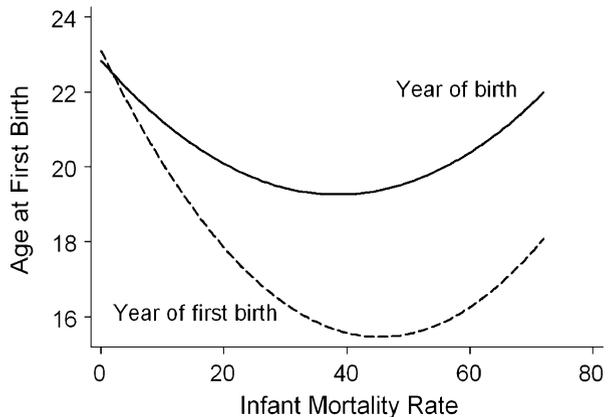
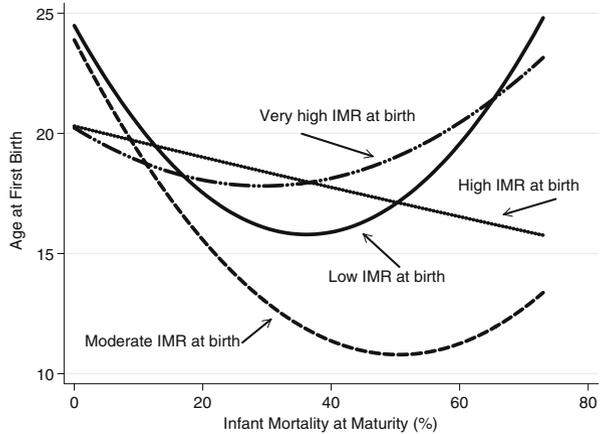


Fig. 4 Interaction effects of IMR at maturity and in infancy on age at first birth ($R^2=0.37$)



somewhat stronger predictor of RS (9%) compared with IMR in infancy (7%). The interaction term for IMR at birth and maturity was not significant and is not shown. The non-significant interaction suggests that effects of mortality on fertility in early and later development are additive.

Year of birth is a reasonable control on RS; however, since some women in the analysis had not completed reproduction by the year 2000, a model for censored data is appropriate. Furthermore, given low counts and standard deviations for RS, Poisson regression is the preferred analysis. Censored Poisson regression (CPOISSON) for RS in STATA 10 (last two columns in Table 5) gave the same signs for effects and somewhat lower *p*-values than the OLS estimates for all coefficients except for the interaction term, which had a higher (non-significant) *p*-value. In sum, Censored Poisson regression does not change the conclusions presented here.

Discussion and Conclusions

Results show significant effects of extrinsic mortality in infancy and at maturity on reproductive outcomes. Main effects at maturity show somewhat, but not substantially, stronger effects on later reproduction. These dual effects suggest multiple pathways for reproductive outcomes with potentially important mechanistic differences. Furthermore, analysis of interactions between early and later environment indicates important developmental tuning for age at first birth: When early conditions are extremely harsh,

Table 4 Mean of IMR at birth (%) intervals used in analysis of interactions in Fig. 3

IMR at birth	OLS coefficient	OLS mean
Low IMR (OLS Constant)	7.55	7.55
Moderate IMR	8.99	16.54
High IMR	22.66	30.21
Very high IMR	49.20	56.75

Table 5 Multiple linear regression showing effects of IMR on RS

RS	B	<i>p</i>	r_p^2	R_p^2 quadratic IMR effects	Censored Poisson	
					B	<i>p</i>
(Constant)	47.66	0.42			35.39	0.00
Year of birth	-0.02	0.44	0.00		-0.02	0.00
IMR at birth	0.11	0.02	0.05		0.03	0.00
IMR at birth squared	-0.001	0.07	0.03	0.07	-0.003	0.04
IMR at first birth	0.16	0.02	0.04		0.03	0.01
IMR at first birth squared	-0.002	0.02	0.04	0.09	-0.005	0.01
		$R^2=0.16$				

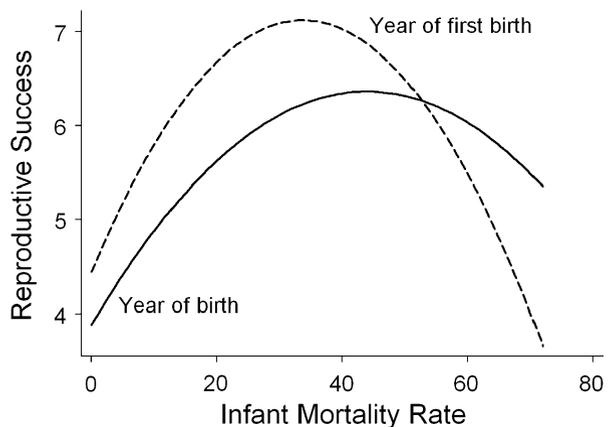
B = unstandardized regression coefficient; *p* = *p*-value for unstandardized regression coefficient; r_p^2 = partial Pearson's correlation coefficient squared; the fourth data column shows coefficient of determination for quadratic effects; Censored Poisson B is the regression coefficient for a right-censored Poisson regression; Censored Poisson *p* is the *p*-value for the Poisson regression

later conditions are less influential in shaping reproductive behavior. Regression models, however, indicated substantial unexplained variance.

Lack of control for individual and household economics is an important limitation of the study. Given the historical nature of the demographic data, adequate economic controls are not possible because it would be impossible to reconstruct reliable economic indicators contemporaneous with events occurring as many as 75 years in the past. Because of secular trends, individual and household economics may be correlated with population IMR, and those correlations could mediate effects shown here. However, mediation (if it exists) would indicate a socioeconomic pathway for the effects of extrinsic risk on life history and would not invalidate results presented here.

There are several important obstacles in studying human extrinsic mortality. The difficulty in disaggregating intrinsic and extrinsic components of mortality is an important consideration for any study of extrinsic risk, especially because intrinsic and extrinsic risk are predicted to have different effects on reproductive strategies. Ideally we would like to measure the effects of mating effort and parenting effort on

Fig. 5 Effects of IMR in infancy and at maturity on pace of reproduction (RS adjusted for age; $R^2=0.16$)



mortality; then extrinsic mortality would be captured in the residual variance (see Quinlan 2006). Unfortunately, such an analysis is virtually impossible here. A somewhat more tractable approach, but still a daunting task, would be to identify all *within*-population effects on mortality; then the residual variance would be a reasonable proxy of extrinsic mortality. That residual variance could then be used as a variable in a *between*-population analysis of extrinsic mortality and reproductive strategies. Obviously, such a study would be a huge undertaking, and the comparability of data across populations would be questionable. A historical analysis, such as this study of rural Dominica, is one approach to developing an empirical corpus to test life history predictions. I suspect that in this study there is some within-population and within-cohort variance in mortality, but the high to extremely high levels of infant mortality suggest substantial extrinsic (inescapable) risk of death. Populations with more modest levels of variation in mortality are probably less useful for historical analysis. Similarly, cross-cultural analyses that capture large variance in mortality may be useful for exploring extrinsic risk effects on life history (e.g., Walker et al. 2006). The empirical situation is tolerable at this early stage of development, but eventually higher-resolution analyses will be required.

Psychological Mechanisms for Human Life History Variation

A handful of studies show effects of mortality on human reproductive behavior, but proximate mechanisms are not well known. Other research suggests that parental responsiveness, mediated by attachment and stress hormones, may play an important role in human reproductive development (Flinn et al. 2008). In the parental responsiveness model, parents respond to environmental risks by altering parenting behaviors in ways that could enhance fitness (Quinlan 2007). A low parental effort strategy, in turn, could induce in offspring a sense that the environment is risky and that development toward a low parental effort strategy would be adaptive (Belsky et al. 1991). However, results presented here indicate that long-term developmental trajectories are not the only factors influencing reproductive strategies—IMR at maturity had a somewhat (though not substantially) stronger effect on reproductive behavior. This finding suggests parallel paths to reproductive strategies: a developmental path responding to early environmental conditions, and (perhaps) physiological (Vitzthum 2001) and conscious decision paths responding to later conditions. Interactions between early and later environmental conditions on reproductive strategies suggest further intriguing possibilities. Under especially harsh conditions at birth, extrinsic mortality later in life had little effect on age at first birth. Other research has shown that harsh conditions in the first year of life had important developmental consequences (Rutter et al. 2007). Effect on reproductive behavior may be due to somatic restrictions on reproductive development requiring more time to accrue necessary somatic resources to begin reproducing (Ellis 2004).

Whatever the mechanisms involved, levels of extrinsic risk may lead to risk-sensitive adult phenotypes involving a broad suit of personality and behavioral traits (Chisholm 1999a; Quinlan and Quinlan 2007; Rutter and O'Connor 2004; Schechter and Francis 2010). Life history strategies might be encoded as attachment and personality traits targeting adaptive motivations. Environmental deprivation in infancy, for example, has been associated with disinhibited attachment characterized

by rapidly developing and shallow bonds (O'Connor et al. 1999) that could support mating effort behaviors later in life.

Other personality facets seem well-suited for regulating reproductive effort. For example, locus of control (LOC) is a psychological measure of an individual's perception of their control over events. People with an external locus of control believe that they cannot influence outcomes through their own efforts—they perceive extrinsic risk. Studies in Western cultures show that external LOC was associated with teen pregnancy (Young et al. 2001, 2004), “risky” sexual behavior (Loue et al. 2004), externalizing behaviors (Jackson et al. 2000), and positive attitudes toward promiscuous sexuality (Werner-Wilson 1998)—traits indicative of mating effort strategies.

Time perspective has also been linked with reproductive strategies. Differences in time perspective are predicted to target potential payoffs to current versus future reproduction (Chisholm 1999b). “Present orientation”—or focus on current rewards rather than delayed gratification—is associated with current reproduction and a mating effort strategy. Conversely, “future orientation”—or focus on delayed gratification—is associated with delayed reproduction and a parenting effort strategy (Chisholm 1999b). Present orientation has been associated with impulsivity and risk-taking behaviors (Robbins and Bryan 2004) and is associated with risk perception (Schechter and Francis 2010).

Impulsivity is also correlated with aspects of mating effort strategies in rhesus monkeys (Gerald et al. 2002) and humans (Comings et al. 2002; Donohew et al. 2000; Hoyle et al. 2000; Zuckerman and Kuhlman 2000). Impulsivity may underlie mating effort behavioral strategies, which often include “present orientation” or lack of willingness to delay gratification (Chisholm 1999a) and externalizing behavior such as delinquency (Belsky et al. 1991). Impulsivity, in fact, has been associated with multiple risk-taking behaviors (Aklin et al. 2005). Development of impulsivity has been linked to unstable family environments during childhood (Elder et al. 1986).

Facets of the Big Five or Five-Factor Model of personality may also play a role in life history strategies. Several studies show that domains of the Five-Factor Model are related to risk proneness and sexual behavior. Low levels of Agreeableness and Conscientiousness, and high levels of Neuroticism, are associated with unsafe sex and intravenous drug use (Trobst et al. 2002). Excitement-seeking is a facet of Extraversion similar to sensation-seeking (Costa and McCrae 1992) which may predispose individuals higher in Extraversion to engage in mating effort strategies. And impulsivity is a facet of Neuroticism (Costa and McCrae 1992), which may also contribute to mating effort strategies.

Personality may respond to and provide a kind of psychic scaffolding for cultural models related to environmental risk and life history strategies. Clear cross-cultural variation in cultural models related to locus of control, impulsivity, time perspective, and so on, are associated with parenting practices (Quinlan and Quinlan 2007) that, in turn, are associated with extrinsic mortality (Quinlan 2007). This line of research, though only suggestive at present, indicates enormous potential for a life history perspective in accounting for important aspects of human cultural diversity—a long-standing goal of evolutionary anthropology.

In sum, a small body of research suggests an important role for extrinsic risk in shaping human reproductive behavior. This study indicates that extrinsic risk early in

development and later in life is significantly associated with female reproductive behavior. Further research should begin to focus on better measures of extrinsic risk, mechanisms for the development of human reproductive behavior, and broader aspects of risk-sensitive phenotypes, including personality and cultural cognition.

Acknowledgments Thanks to the Saint Sauveur Roman Catholic Church for access to St. David Parish historical birth and death records. Thanks to friends in Bwa Mawego (too many to mention here) for their tremendous help with local oral histories and ethnographic interviews. Thanks to Heather Bonander for research assistance with the historical demographic data. Thanks to the Central Statistics Office of the Commonwealth of Dominica for research permission. Thanks to Drs. Marsha Quinlan and Mark Flinn for constant collegial support. This research was funded in part by NSF Cultural Anthropology grant BCS-0650317.

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