

# Extrinsic mortality effects on reproductive strategies in a Caribbean community

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June 30, 2009

Presented at EAS/AAA 2008, Submitted to *Human Nature*

## **Abstract**

Extrinsic mortality is a key influence on organisms' life history strategies, especially age at maturity. This study examines effects of extrinsic mortality through the life course on human age at maturity and pace of reproduction. Extrinsic mortality indicated by local population infant mortality rates (IMR) between years 1925 and 2001 in infancy and at maturity show effects on age at first birth and pace of reproduction among 125 women in rural Dominica. Parish death records show huge historical variation in age specific mortality rates. Infant mortality in the early 1920's was low, increases dramatically beginning in 1929 and reaches a maximum in the 1950's, at which point IMR declines steadily to its present low rate. Mortality rate in early life showed a quadratic association with age at first birth. Under low mortality women reproduced relatively late in life. At moderately high levels of mortality women tended to reproduce earlier than at 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 conditions leading 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 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 in infancy, extrinsic mortality later in life had little effect on adult

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

**Keywords:** Risk; teen pregnancy; child development; evolutionary ecology; behavioral ecology; demography; personality.

## **Introduction**

Extrinsic mortality is the risk of death that is not conditional on an organism's reproductive behavior (Stearns 1992:182). 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. Extrinsic mortality plays a key role in the evolution of life histories and reproductive strategies (Stearns 1992; Roff 2002; Chisholm 1993, 1999a; Promislow & Harvey 1990). When extrinsic mortality is high, then organisms should reproduce relatively early in life to reduce mortality exposure over 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 with juvenile mortality negatively correlated with age at maturity, age at weaning, maternal investment, and positively correlated with litter size, and pace of reproduction (Promislow and Harvey 1990: 424).

For humans extrinsic risk has attracted theoretical interest since the early 1990s (e.g. Borgerhoff Mulder 1992; Chisholm 1993, 1999a; Harpending, Draper & Pennington 1990); however, empirical work is relatively scarce. Several studies show predicted relations between extrinsic risk and human life history patterns. Mortality was negatively associated with age at reproductive maturity among urban Americans (Wilson & Daly 1997), Sub-Saharan Africans (Gant, Heath, & Ejikeme 2006) and in two cross-national studies (Low et al. 2008; Anderson n.d.). Extrinsic risk predicts patterns of parental care cross-culturally (Quinlan 2007; Quinlan 2006). And perception of mortality may influence human reproductive behavior (Chisholm et al. 2005). Even in this small body of research 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, has been suggested as a sensitive period for development that has strong effects on adult outcomes (e.g. Belskey et al. 1991; Chisholm 1999a; Draper & Harpending 1982; Ellis et al. 1999; Quinlan 2000; Quinlan & Quinlan 2007). In these related early childhood models, parental 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 and associated hormones have been implicated (Ellis 2004; Flinn et al. 2008). The parental responsiveness-early childhood theory of reproductive development suggests strong vertical or intergenerational transmission of adult phenotypes (Quinlan & 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 (Whiting 1980; Quinlan & Flinn 2003). 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 adolescences (Quinlan 2000). 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 conscious decision making is likely important.

The present study cannot address mechanistic questions; however, the study focuses on timing of environmental cues by examining effects of population-level extrinsic mortality at birth and age of maturity on timing of first birth and pace of reproduction in an extraordinarily variable environment in a rural community in the Commonwealth of Dominica. The study takes advantage of a naturalistic experiment occurring between 1925 and 2001 to examine population extrinsic mortality rates in year of birth and year of 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 is predicted to show quadratic associations with reproductive behavior (see fig 1).

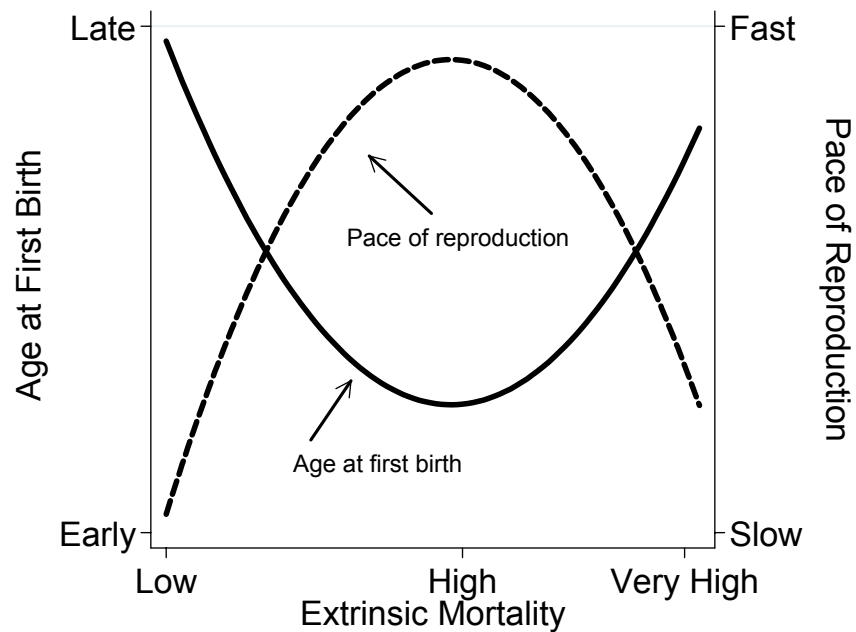


Fig. 1. Predicted relations between intrinsic mortality and reproductive strategies.

### The Site

Large variation in historical infant mortality rates in rural Dominica present an instructive natural experiment in 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. There are about 700 full and part-time residents. Currently average annual household income in Bwa Mawego is approximately \$5,000 E.C. (\$1,850 U.S.). 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*, currently is the most 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 in the recent past conditions were much worse. Currently the infant mortality rate (IMR) for the island is 17 per 1000 live births compared to 46 per 1000 for the Caribbean region (U. S. Census Bureau [www.census.gov/ipc/www/idbnew.html](http://www.census.gov/ipc/www/idbnew.html)); however, IMR for St. David Parish reached a remarkable and tragic high of 724 per 1000 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 9<sup>th</sup> and 10<sup>th</sup> grade 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 & Flinn (2005), and Quinlan (2006).

## **Methods**

Infant mortality rates (IMR) indicating extrinsic risk were calculated from St. David Parish birth and death records for years 1925 through 2001. Here IMR is defined as deaths by age 1 year divided by total births for the year. 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. Parish death records show little evidence of reporting biases: The handwriting is consistent over many years and changes in hand writing are not associated with substantial changes in IMR, indicating a single recorder of vital events for the Parish over 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. 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 (during gestation and early infancy); IMR in the year the woman gave birth is 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 linear function of age (quadratic terms were not statistically significant and inclusion of quadratic terms did not appreciable change the shape or slope of the RS-maternal age function).

Analyses presented used OLS multiple linear 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, range and standard deviation 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; hence, OLS results are presented here because OLS coefficients give a more intuitive sense of the outcomes. Otherwise the models conform to the assumptions of OLS regression. Alpha was set at .05 for first order effects, and .10 for quadratic and interaction effects.

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

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

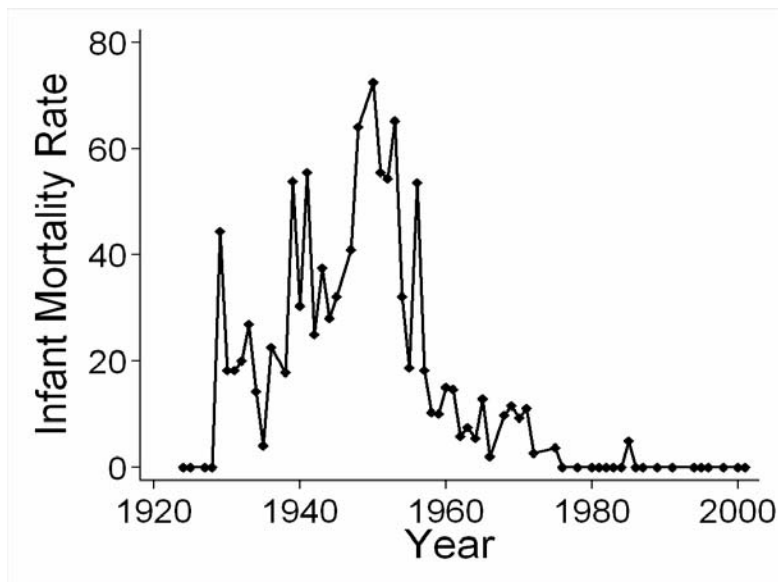


Fig. 2. Historical trends in infant mortality in rural Dominica.

### 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 1920's show huge variation in age specific mortality rates. Infant mortality in the early 1920s was low, but IMR increases 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 (which occur regularly in Bwa Mawego) exacerbated by deteriorating economic conditions (Honeychurch 1995:160-1) is one likely explanation. Infant mortality rates were high and variable from 1929 throughout the 1950s (fig. 2); however, rates are 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 post-war reconstruction. Locals recount an extreme shortage of meat (usually imported) and fish due 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 (*Ibid.*). 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 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 1990’s many villagers I knew said it was illegal to keep pigs in the village, and 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 ( $r^2 < .01$ ), 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 ( $r = -.26$ ) with substantial “unaccounted for” variance due to the variability in IMR. IMR at maturity and year of birth showed a more substantial negative correlation ( $r = -.69$ ) indicating the general downward trend in population IMR after 1950. This latter correlation suggests the possibility of problematic multicollinearity in the regression models. However, the maximum Variance Inflation Factor (VIF) for models presented in tables 3

and 4 (after removing quadratic effects, which inevitably result in substantial VIFs) was 2.5, indicating that multicollinearity was not a problem in these models.

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

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

Population IMR during infancy showed a quadratic association with age at first birth table 3, figure 3. Under 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 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).

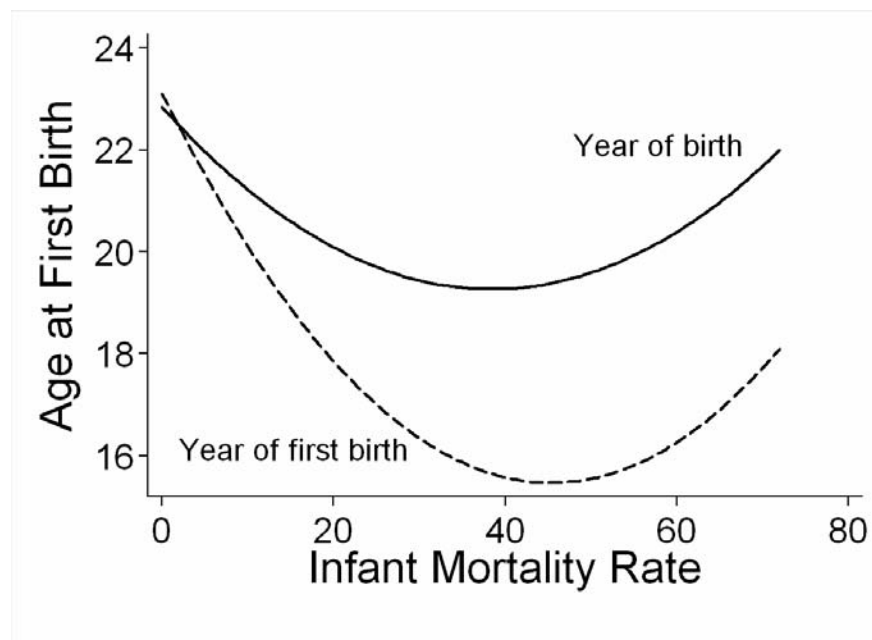


Fig. 3 Effects of IMR in infancy and at maturity on Age at first birth

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

<b>AFB</b>	<b>B</b>	<b>p</b>	<b>r<sub>p</sub><sup>2</sup></b>	
(Constant)	601.15	0.00		
<b>Year of birth</b>	-0.30	0.00	0.31	
<b>IMR at birth</b>	-0.19	0.00	0.07	
<b>IMR at birth squared</b>	0.002	0.01	0.05	0.13
<b>IMR at first birth</b>	-0.34	0.00	0.11	
<b>IMR at first birth squared</b>	0.004	0.00	0.07	0.19
<b>IMR X IMR</b>	0.004	0.01	0.05	0.05

**R<sup>2</sup>**= 0.68 0.37

IMR<sub>x</sub>IMR with highest value Winsorized to the next highest value yielded r<sub>p</sub><sup>2</sup>=0.04 other effects were basically unchanged.

There were complex interactions between population mortality rates in infancy and at maturity in the effect on age at first birth indicated in figure 4. The graphic representation in figure 4 should be taken as an approximation of the interaction 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 was low to moderate in infancy, extrinsic mortality later in life had significant effects on adult reproduction.

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

<b>IMR at Birth</b>	<b>OLS Coefficient</b>	<b>OLS Mean</b>
<b>Low IMR (OLS Constant)</b>	7.55	7.55
<b>Moderate IMR</b>	8.99	16.54
<b>High IMR</b>	22.66	30.21
<b>Very High IMR</b>	49.20	56.75

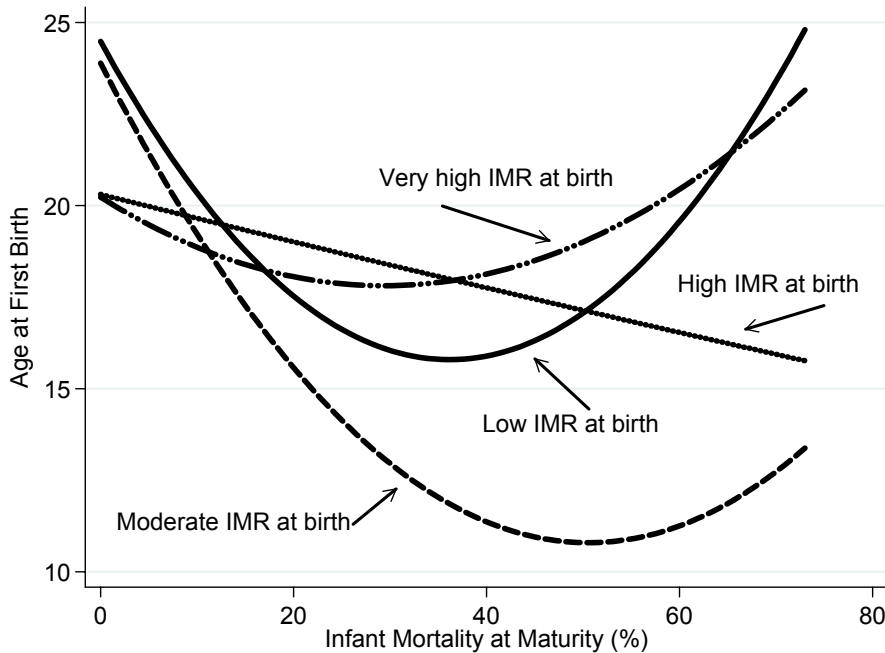


Fig. 4 Interaction effects of IMR at maturity and in infancy on age at first birth.

The pace of reproduction estimated as surviving offspring controlled for maternal age showed a similar, but inverted, quadratic effect (table 5, fig. 5). Pace of reproduction increased as IMR increased, but decreased at very high mortality levels. No doubt, the strong downward trend in RS at IMR rates above 40% reflect 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 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 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.

Given low counts and standard deviation for RS, Poisson regression is the preferred analysis. Multiple Poisson regression for RS (not shown) gave the same signs for effects and somewhat lower p-values than did OLS for all coefficients except for the interaction term which had a higher (non-significant) p-value. Psuedo-R-squared for Poisson regression was lower (.09) than R-squared for OLS regression (.16). In sum, Poisson regression does not change the conclusions presented here.

Table 5. Multiple linear regression showing effects of IMR on RS

RS	B	p	$r_p^2$	
(Constant)	47.66	0.42	0	
<b>Year of birth</b>	-0.02	0.44	0.00	
<b>IMR at birth</b>	0.11	0.02	0.05	
<b>IMR at birth squared</b>	-0.001	0.07	0.03	0.07
<b>IMR at first birth</b>	0.16	0.02	0.04	
<b>IMR at first birth squared</b>	-0.002	0.02	0.04	0.09
	<b>R<sup>2</sup>=</b>		0.16	0.16

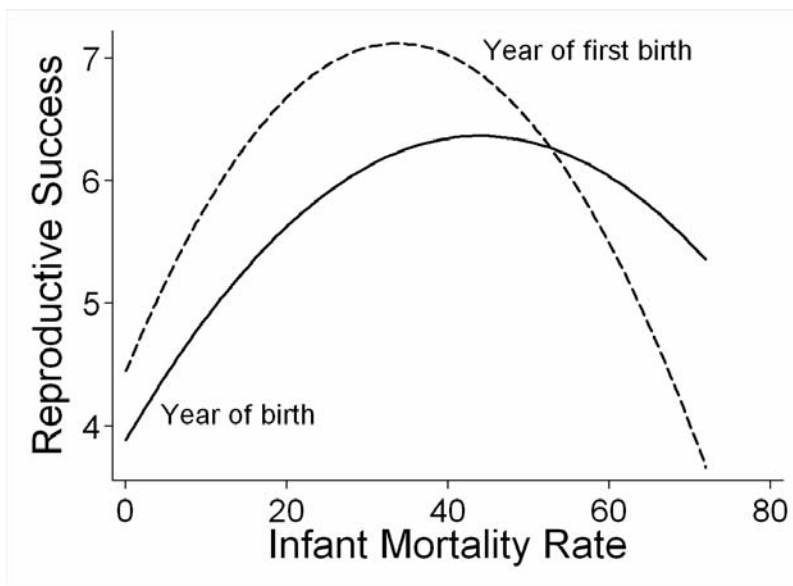


Fig. 5 Effects of IMR in infancy and at maturity on pace of reproduction (RS adjusted for age).

## **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 two pathways for reproductive outcomes with potentially important mechanistic differences. Furthermore, analysis of interactions between early and later environment indicate important developmental tuning for age at first birth: When early conditions are extremely harsh, then later conditions are less influential in shaping reproductive behavior.

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.

Proximate mechanisms by which extrinsic mortality affects reproductive behavior cannot be indicated in this study. Other research suggests that parental responsiveness, mediated by 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 a (perhaps) conscious decision path 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. This effect 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). Life history strategies might be encoded as personality traits targeting adaptive motivations. 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. 2004; Young, Martin, Young & Ting 2001), “risky” sexual behavior (Loue et al. 2004), externalizing behaviors (Jackson, Frick & Dravage-Bush 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 (*Ibid.*). Present orientation has been associated with impulsivity and risk taking behaviors (Robbins & Bryan 2004).

Impulsivity is also correlated with aspects of mating effort strategies in humans (Comings et al. 2002; Donohew et al. 2000; Hoyle, Fejfar & Miller 2000; Zuckerman & Kuhlman 2000) and rhesus monkeys (Gerald et al. 2002). Impulsivity may underlie such behavioral strategies, which often include “present orientation” or lack willingness to delay gratification (Chisholm 1999) and externalizing behavior such as delinquency (Belsky, Steinberg & Draper 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, Caspi & Downey 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, Conscientiousness and high 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 & McCrae 1992) that may dispose individuals higher in Extraversion to engage in mating effort strategies. And impulsivity is a facet of Neuroticism (*Ibid.*), 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 etc. 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, though largely abandoned 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 mechanisms for the development of human reproductive behavior and broader aspects of risk sensitive phenotypes including personality and cultural cognition.

### **Acknowledgements**

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 Drs. Marsha Quinlan and Mark Flinn for constant collegial support in the field and at home.

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