

# UC Davis

## UC Davis Previously Published Works

### Title

Demographic consequences of unpredictability in fertility outcomes

### Permalink

<https://escholarship.org/uc/item/6rj992v7>

### Journal

American Journal of Human Biology, 14(2)

### ISSN

1042-0533

### Authors

Leslie, Paul  
Winterhalder, Bruce

### Publication Date

2002-03-01

### DOI

10.1002/ajhb.10044

Peer reviewed

## Demographic Consequences of Unpredictability in Fertility Outcomes

PAUL LESLIE<sup>1,2,3\*</sup> AND BRUCE WINTERHALDER<sup>1,2</sup>

<sup>1</sup>*Department of Anthropology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina*

<sup>2</sup>*Curriculum in Ecology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina*

<sup>3</sup>*Carolina Population Center, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina*

**ABSTRACT** Child survival is probabilistic, but the unpredictability in family formation and completed family size has been neglected in the fertility literature. In many societies, ending the family cycle with too few or too many surviving offspring entails serious social, economic, or fitness consequences. A model of risk- (or variance-) sensitive adaptive behavior that addresses long-term fertility outcomes is presented. The model shows that under conditions likely to be common, optimal, risk-sensitive reproductive strategies deviate systematically from the completed family size that would be expected if reproductive outcome is were predictable. This is termed the “variance compensation hypothesis.” Variance compensation may be either positive or negative, resulting in augmented or diminished fertility. Which outcome obtained is a function of identifiable social, economic, and environmental factors. Through its effect on fertility behavior, variance compensation has a direct bearing on birth spacing and completed fertility, and thereby on problems in demography and human population biology ranging from demographic transitions to maternal depletion and child health. Risk-sensitive models will be a necessary component of a general theory of fertility. *Am. J. Hum. Biol.* 14:168–183, 2002. © 2002 Wiley-Liss, Inc.

Child bearing and family building are subject to myriad uncertainties. This unremarkable claim will surprise few demographers, anthropologists, or parents, yet the consequences of unpredictability in vital events and family formation have remained marginal to mainstream demography. Little is known about how unpredictability is perceived and how it affects behavior (consciously or unconsciously). In this paper, it is argued that unpredictability is of importance in understanding a variety of demographic problems.

The implications of unpredictability in family building (reproductive outcomes) for fertility and family demography are explored by means of a mathematical model. The model represents unpredictability in outcome primarily in terms of child survival. The approach is grounded in neo-Darwinian theory (Winterhalder and Smith, 1992; Smith and Winterhalder, 1992) and is an outgrowth of general work in evolutionary ecology on risk-sensitive adaptation (Winterhalder et al., 1999). Although couched in neo-Darwinian terms, the implications of “variance compensation” extend to any orientation or discipline (e.g., human

biology, economics, sociology) that takes human fertility to be a salient problem. Two related questions are addressed: (1) How might individuals, couples, or families respond rationally (adaptively) to unpredictability in family building? (2) What are the implications of these responses for fertility at the population level, particularly for demographic transitions?

### SOURCES OF VARIATION AND UNPREDICTABILITY

Various stochastic processes contribute to the overall unpredictability in producing a descending generation of a desirable size and composition. There is unpredictability in the supply (production) of children arising from factors as diverse as variation in fecundability, fetal loss, contraceptive failure, and death of a spouse (Wood, 1994; Bongaarts, 1984). Child survival is critical, and is the specific focus of our model. In

\*Correspondence to: Paul Leslie, Carolina Population Center, 123 West Franklin Street, Chapel Hill, NC 27516.  
E-mail: pwleslie@unc.edu

Received 13 September 2000; Revision received 4 May 2001; Accepted 5 October 2001

addition, children may survive to adulthood yet fail to fulfill the utility expectations of their parents, either because of lack of ability or because they abdicate on social responsibilities to the ascending generation. Uncertainties are compounded when the sexual composition of a sibling set—itself subject to chance—is important (Leslie and Dyson-Hudson, 1999).

Demography has been concerned with unpredictability of child survival primarily because of its methodological rather than behavioral implications. Heterogeneity in the causes of mortality creates within- and between-family variance in numbers of children dying (Gardiner and Yerushalmy, 1939); this is either treated as a potentially serious methodological problem (Trussell and Rodriguez, 1990) or is seen as an opportunity to gain further knowledge of the determinants of mortality (Zaba and David, 1996; Ronsmans, 1995; Das Gupta, 1990). This work reflects an implicit assumption that once the biological or socioeconomic factors thought to affect mortality have been accounted for, residual variation is “noise,” an analytic irritant not containing useful information or adaptive consequences.

Heterogeneity is viewed here differently. From a behavioral ecology perspective, unpredictable variation in fertility outcome is more than a methodological nuisance or an empirical opportunity to identify the determinants of mortality. It is a reality with important consequences for behavior. Mortality is undeniably relevant to fertility and family building, but the unpredictable variance in mortality outcomes is just as important as the average loss of children. To demonstrate this, a risk-sensitive model of fertility is developed, how this approach differs from past treatments of unpredictability of family building in the demographic literature is specified, and the relevance of this view for understanding the persistent problem of demographic transitions is explored.

#### CONCEPTUAL ELEMENTS OF A RISK-SENSITIVE MODEL

In a deterministic world, family building outcomes would be fully predictable. A couple would know how many additional births they should plan in order to compensate with assurance for offspring who die. However, in the world of experience,

long-term reproductive success and family composition are to some degree unpredictable. Because of factors that include stochastic mortality, the outcome of fertility behavior during a couple's reproductive span is realized at its conclusion as odds over a range of completed family sizes. This means that income, economic security, social status, genetic fitness, and other metrics that vary with completed family size are similarly afflicted with unpredictability.

In reality, offspring survival to adulthood is a probability density function with a range from 0 to the number of births ( $b$ ). In this circumstance, a couple's best reproductive strategy depends jointly on the consequences (costs) of overshooting or undershooting the target family size by varying amounts, and on the probabilities of those outcomes. It is this compound set of relationships that is modeled and explored. The important general point is that stochasticity itself has a bearing on the best-choice reproductive strategy that is analytically separable from that of average outcomes and predictable losses.

#### *Key concepts and definitions: Risk and value*

The term “risk” is used in the standard economic sense of unpredictable variability in an outcome that has economic, adaptive, or other significance to the actor. This use differs from the epidemiological (and colloquial) usage that equates risk with degree of peril or exposure to hazard. Risk is also distinct from “uncertainty,” which refers to incomplete knowledge of outcome probabilities (Friedman et al., 1994). From the point of view of the actor, outcomes may be unpredictable either because they are fundamentally stochastic or because information about determinants of outcomes is unavailable or too costly to obtain. When the consequences of a behavior such as fertility are unpredictable to some degree, they are best described in terms of an *outcome distribution*.

“Value” is a measure of the relative desirability of outcomes. A working assumption in both behavioral ecology and microeconomic theory is that people tend to select behaviors with outcomes that maximize value (“utility” in economics, “fitness” in evolutionary biology), given applicable constraints (Foley, 1985; Elster, 1986). The choice of a particular metric or currency is an important problem in its own right, but

we purposefully avoid specification. Rather, we use the term *value* as a gloss for any appropriate metric, in order to emphasize the generality of the model and its implications. The relationship between outcome (number of surviving offspring) and the payoff to that outcome is described by a *value function*.

The logical structure of the model is thus: (a) couples or other decision-making units choose from among a constrained set of behavioral alternatives (number of births); (b) those choices result in an outcome distribution of completed family sizes; (c) each outcome has an associated value which allows one to identify the optimum from among the original alternatives (Stevens and Charnov, 1982; Winterhalder et al., 1999).

#### *Outcome distributions*

Despite unpredictability, the hypothetical couple must settle on the best number of children to bear. More specifically, what number of births ( $b$ ) will, when realized as a completed, adult sibling group (size,  $s$ ), generate the highest expected value,  $E[U(b)]$ ? If there is no childhood mortality, the optimum number of births ( $b$ ) is equal to the outcome for which the utility of surviving offspring,  $U[s]$ , is greatest. If there is deterministic mortality in which all couples invariably experience the same loss, and if the probability of surviving childhood is  $p$ , then a couple can compensate reliably by having  $b^*/p$  births, where  $b^*$  is the optimum number of births if all survive childhood. More realistic is the case where each birth has a certain stochastic probability of surviving. The outcome ( $s$ , number of children surviving to adulthood) for a given number of births ( $b$ ) can be described by a binomial distribution. That is,

$$f_b(s) = C[b, s]p^s(1-p)^{b-s} \quad (1)$$

where  $C[b, s] = b!/[s!(s-b)!]$  and  $p$  is the probability of offspring survival. In a stochastic world, births translate into a range of completed family size outcomes according to binomial odds.

#### *Value functions*

For humans and other species, the value function is unlikely to be linear over the range of possible number of offspring. This

is so for several reasons. The value of a child depends critically on the number and characteristics of other offspring, interacting with the family's mode of production, resources, socioeconomic context, etc. For instance, adding a second child to a family's labor pool may have a much greater, positive impact on agricultural productivity than adding an eighth child, in which case the rate of increase of the value function will decrease with parity. The costs of having children, whether measured in terms of economics or health, also are likely to be nonlinear functions of number of offspring and may at some point in a growing offspring cohort outweigh the incremental benefits of additional children. The total value of all children will then begin to decline, giving the value function an intermediate peak. The reader will easily envision other possibilities.

#### *Risk-sensitive analysis*

If fertility outcomes are fully predictable, the highest point of the value function indicates in a straightforward way the optimum reproductive behavior. This is true whether the value function is linear or nonlinear. Conversely, in a stochastic system, unpredictability in outcome has no bearing on the optimal behavioral choice if the value function is linear. With a linear value function, symmetrical outcome dispersion above and below the mean perfectly cancel one another; the mean alone is sufficient to rank outcomes. More interesting—and we believe much more common—are situations in which outcomes are unpredictable *and* the value function is nonmonotonic (nonlinear). This is the realm of risk-sensitive analysis.

To illustrate, assume that under a certain set of socioeconomic conditions, the optimum completed family size (number of surviving offspring,  $s^*$ ) is four. Ending the family cycle with fewer or more than four adult offspring entails costs, with particularly severe consequences associated with two or fewer. The values of these outcomes [ $U(s)$ ] relative to the optimum [with  $U(s^*) = 1$ ], for  $b = 0-10$  births, are shown in column (b) of Table 1. The same values are represented graphically in Figure 1a. The numbers are hypothetical, but we argue that the general shape of this function is likely to be appropriate for many human populations (Winterhalder and Leslie, 2002). Figure 1a is dubbed a

TABLE 1. Distribution of outcomes and expected values of alternative numbers of births, with probability of survival 0.75

(a)	(b)	(c) 4 births		(d) 5 births		(e) 6 births		(f) 7 births		(g) 10 births	
Outcome (no. of grown children)	Value	Likelihood (binomial)	Weighted value	Likelihood (binomial)	Weighted value	Likelihood (binomial)	Weighted value	Likelihood (binomial)	Weighted value	Likelihood (binomial)	Weighted value
10	0.816	—	—	—	—	—	—	—	—	0.056	0.046
9	0.872	—	—	—	—	—	—	—	—	0.188	0.064
8	0.918	—	—	—	—	—	—	—	—	0.282	0.258
7	0.954	—	—	—	—	—	—	0.133	0.127	0.250	0.239
6	0.980	—	—	—	—	0.178	0.174	0.311	0.305	0.146	0.143
5	0.995	—	—	0.237	0.236	0.356	0.354	0.311	0.310	0.058	0.058
4	1.000	0.316	0.396	0.396	0.396	0.297	0.297	0.173	0.173	0.016	0.016
3	0.860	0.422	0.363	0.264	0.237	0.132	0.113	0.058	0.050	0.003	0.003
2	0.462	0.211	0.097	0.088	0.041	0.033	0.015	0.012	0.005	0.000	0.000
1	0.078	0.047	0.004	0.015	0.001	0.004	0.000	0.001	0.000	0.000	0.000
0	0.000	0.004	0.000	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Expected value			0.78		0.90		0.95		0.97		0.92

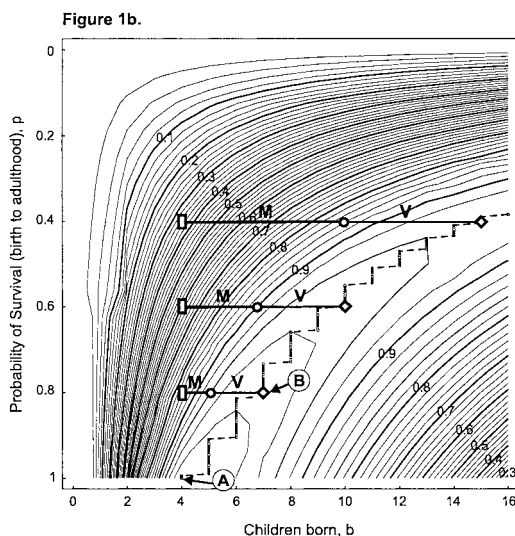
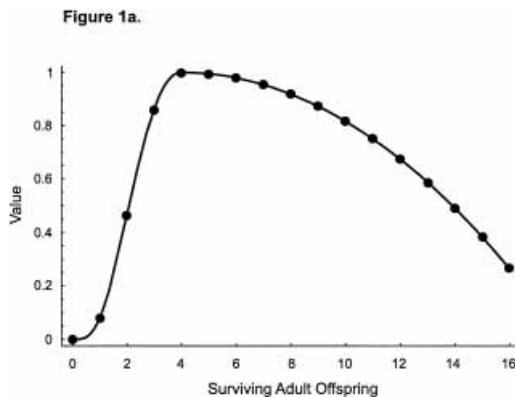


Fig. 1. (a) Proposed right-ibis value function for number of adult offspring, (b) Iso-value surface based on the value function shown in (a) and a binomial outcome distribution for survivorship of children. Contour lines connect points of equal utility □: optimum number of births with no mortality ( $p = 1$ ); ○: optimum number of births with “deterministic” mortality ( $p < 1, \sigma^2 = 0$ ); ◇: optimum risk-sensitive number of births [ $p < 1, \sigma^2 = p(1-p)n$ ]; M = extra births needed to offset the mean effect of mortality; V = additional compensation needed to offset stochastic variance.

“right-ibis” value curve, after the right-facing profile of the long-billed shore bird; its mathematical formulation is set out in the Appendix.

Table 1 shows the expected values of a sample of alternative reproductive choices, given the value function described by Figure 1a and column (b) of the table. It also gives (columns c-g) the likelihood for each

outcome possible for a given number of births, (binomial odds, probability of survival  $p = 0.75$ ), and the weighted value of that outcome. The overall value of  $b$  births is equal to the sum of the values of all possible outcomes, each outcome weighted by its likelihood:

$$E[U(b)] = \sum_s U(s)f_b(s) \quad (2)$$

where  $U(s)$  is the value of  $s$  surviving offspring,  $f_b(s)$  is the probability function specific to  $b$  births, and the sum ( $\Sigma_s$ ) is over values of  $s$  from 0 to  $b$ .

A completed family of four surviving children ( $s = 4$ ) has the highest possible value. However, at 25% mortality ( $p = 0.75$ ), a couple having four births would actually end up with four surviving children less than a third of the time (likelihood = 0.316). A surviving sibling set of three offspring is the most common outcome, occurring 42% of the time, and has a relative value of 0.86. Approximately 26% of couples bearing four children would end up with two or fewer surviving offspring, notably inferior outcomes. Overall, the summed value of four births is only 0.78.

Having five births [column (d)] in order to compensate for unpredictable mortality introduces the possibility of ending up with *more* than the ideal number of surviving children. However, because the value function assigns greater costs to undershooting than to surpassing the optimum, the potential cost of too many surviving offspring is more than compensated for by the greatly reduced likelihood of too few survivors. There is now only a 10% chance of two or fewer surviving offspring. The value of this reproductive option increases to 0.90. In fact, the risk-sensitive behavioral optimum is seven births [column (f)], above which value begins to decline. Note also that the alternative that maximizes the probability of achieving the highest utility outcome of four surviving children,  $b = 5$  [column (d)], is nonetheless not the best choice.

#### *An iso-value map for stochastic fertility outcomes*

A continuous, graphic representation of the model shows more clearly the relationships among the model's parameters and

our argument for the potential importance of variance compensation. Figure 1b shows the expected value  $E[U(b)]$  for each possible number of births ( $x$ -axis), for all values of  $p$  (0 to 1;  $y$ -axis), given the value function  $U(s)$  (Fig. 1a). The array of expected values is depicted as an iso-value surface, analogous to a topographic map with contour lines connecting points of equal value rather than equal elevation.

The  $x$ -axis of Figure 1b corresponds to the situation in which there is no unpredictability;  $p = 1$  and all children survive. The iso-value surface at  $p = 1$  is simply a topographic representation of the value function in Figure 1a (note the corresponding peaks at four births). As survivorship decreases (moving up the  $y$ -axis), the unpredictable variance in outcome increases [ $\sigma^2 = bp(1 - p)$ ] and the iso-value contours take the shape of a rightward-curving, declining ridge. More precisely, because children are born as whole (non-fractional) units, the value surface is actually a stepped ridge. This is indicated by the stair-step line depicting the optimal number of births for a given level of mortality. Although it would be more accurate to depict the off-ridge iso-value contours as similar steps, the contours have been smoothed in the interests of clarity. In general, for any given survival probability ( $p$ ), the optimal, risk-sensitive number of births is found by dropping a vertical from the stepped ridge, at that value of  $p$ , to the  $x$ -axis.

The best-choice, risk-sensitive fertility behavior must compensate for more than the average or expected loss of children. When survivorship is unpredictable ( $p < 1$ ) and the value function is nonlinear, the optimal behavior results from both a mean effect and a dispersion or variance (risk) effect. The iso-value map provides a means of separating these effects, best explained by way of example. The optimal family size at  $p = 1.0$  is 4 (point A in Fig. 1b). If  $p$  drops to 0.8, the optimal number of births is 7 (point B in Fig. 1b), an increase of three. Note, however, that only one of these additional births would be needed to offset the mean effect of mortality (80% survival of 5 births yields the optimal 4 surviving children; see segment M in Fig. 1b at  $p = 0.8$ ). The other two births are adjustment for the effect of unpredictable outcome variance (segment V) in combination with an asymmetrical value function. Similar calculations for two

other levels of mortality ( $p = 0.6$  and  $p = 0.4$ ) are also plotted on Figure 1b, showing that the stochastic effect (segments V) can be substantial.

The predicted shift in optimum fertility due to stochastic outcomes is termed the *variance compensation hypothesis* (VCH). For value functions similar to the right-ibis, variance compensation will be positive; unpredictability exerts upward pressure on fertility. This gives the VCH an immediate bearing on a range of problems that involve reproductive tactics and population growth. Value functions with rather different shapes are possible. In particular, value functions that differentially penalize overshooting an intermediate peak can produce *negative* variance compensation (see Winterhalder and Leslie, 2002).

#### RISK-SENSITIVE ANALYSIS OF COMPLETED FERTILITY: THE CASE OF DEMOGRAPHIC TRANSITIONS

##### *The right-ibis value curve: Evidence and rationale*

Theoretical considerations outlined above show that fertility behavior is likely to be risk-sensitive due to stochasticity of outcomes and nonlinear features of the value function. Unfortunately, there is a dearth of information relevant to estimating or constructing empirically based value functions. The large literature on the value of children and related work on ideal family size and sex composition preferences (Lee and Bulatao, 1983; Friedman et al., 1994; Stash, 1996; Hull, 1995) generally is qualitative and not informative about the subtleties of value function shape. Studies that have measured both the economic costs and benefits of children (Cain, 1977; Toulmin, 1992; Hull, 1995; Bock, 1995; Kaplan, 1994) usually deal in averages, perhaps differentiated by sex. Few take number of surviving offspring or even birth order into account. Though a lamentable restriction, until appropriate data have been developed our theoretical purposes will be served if we can establish that a right-ibis value function is conceptually reasonable and perhaps common in well-defined circumstances.

Comments on the negative consequences of raising too few children are commonplace in the family planning and fertility transition literature. For instance, studies in

South Asia show "...instances of misfortune befalling aging parents who do not have a surviving son to depend on... insecurity of property rights, widespread failure of financial and insurance markets, and absence of important extrafamilial welfare institutions—[all of which] point to the salience of parental security concerns for reproductive strategies" (Cain, 1983 p. 691). Difficulties attendant on a surplus of offspring are also evident. When opportunities for employment outside the household or for contributions to subsistence within the household are limited, the marginal value of children is likely to decrease and may be negative above some number. The ability to avoid or mitigate this decline depends on the extent to which diversification or intensification of productive activities is possible. Such elasticity varies greatly among societies and economies (Low et al., 1992; Volland, 1995). Borgerhoff Mulder (1987, 1992) found a marked contrast between two East African populations: Kipsigis farmers suffer from overproducing children because farms cannot be subdivided much further; Datoga pastoralists enjoy greater flexibility.

Studies that focus on the consequences of low completed family sizes tend to ignore the consequences of large families; conversely, discussions of the problems associated with having many children tend to be silent on the consequences of having few. We are left to speculate about cumulative child value on the unexamined end of the outcome distribution. Some exceptions are found in the behavioral ecology literature (Blurton Jones and Sibley, 1978; Blurton Jones, 1987; Boone and Kessler, 1999; Kaplan and Lancaster, 2000; Luttbeg et al., 2000). Here several studies have found that the marginal costs of children for women are U-shaped, or elevated at low and high birth orders (Randall, 1984; Toulmin, 1992). Higher costs at low parities may arise from maternal inexperience or absence of older children who can help with child care. High costs at higher parities may arise from problems of maternal depletion, survival (too many mouths to feed), partible inheritance, or increasing difficulty in achieving or maintaining a desired standard of living. Such costs are a component of value, but do not completely determine a value curve.

Many biocultural and socioeconomic factors can affect the value function. The value of children varies with birth order and

gender in a wide range of societies, past and present. For example, the first-born (or first-born son) may carry value as a sign of fecundity. This may be of great importance to women in some societies, where social status as well as economic security may depend heavily on it. The likelihood of any one offspring marrying, or the desirability of the marriage that can be contracted, may depend on the existence of siblings of certain sex and age. Among the Turkana of northwest Kenya, a herder's children provide security in old age. But their ability to do so depends on their marrying and establishing viable domestic units (Leslie et al., 1999). Bridewealth is very high (often involving hundreds of animals), and bride-wealth received for marriage of one's daughters is a crucial source of wealth for contracting marriage of one's sons. Thus, the ability of sons to get married is constrained by the number of their surviving sisters, which is subject to stochasticity in both survivorship and sex ratio at birth. At very low family size, each added sibling substantially improves the long-term value of the offspring to the parents and the long-term success of the genealogical unit. This type of situation may produce a pronounced convexity (acceleration) of the left side of the value function, like our right-ibis.

Health and demographic considerations are also important. In some populations women suffer from depletion of energy reserves under the stress of repeated pregnancy and lactation (Little et al., 1992; Tracer, 1991; Hobcraft et al., 1985). They express concern over the health consequences of pregnancy and childbirth (Mason, 1997). Short birth intervals, which often contribute to high achieved parity, may compromise the health of both infant and mother (Curtis et al., 1993; Huttly et al., 1992). Such consequences are borne not just by the mother; they may affect her ability to nurture her other children or grandchildren. Biological costs thereby enter the overall calculus, contributing to the right-hand side of the U-shaped relationship between mortality and parity, and to nonlinearity of the value function. Higher mortality may also reflect lower value. Voland (1995) found that mortality of sons in an 18th century German population increased with the number of surviving, older brothers. Parents needed only one heir

and the costs of more sons may have outweighed the benefits, given the limited prospects of the younger sons. Sons in high-ranking families competed with one another for parental resources; marriage rates for third and higher order surviving sons plummeted, perhaps due to selective neglect. This is another way of saying that the value function, at least for sons, decreased at higher completed family sizes. More direct forms of postnatal controls on family size (infanticide, abandonment, fostering), as well as prenatal controls (abortion, terminal abstinence), all constitute evidence that people are aware of and manipulate family size and configuration to adaptive ends.

#### *Brief overview of demographic transition theory*

The term "demographic transition" refers to the shift from a high fertility/high mortality regime to one of low fertility/low mortality. Originally applied to the experience of western Europe during the 19th century, the term has come to embrace similar changes in developing countries during the 20th century. Because of their exceptional importance to issues of population growth, economic development, and environmental quality, demographic transitions have drawn sustained attention from population scholars during the past half century.

Explanation of the decline in fertility has proven much more elusive than understanding the decline in mortality. Early hypotheses (Thompson, 1929; Notestein, 1945; Davis, 1945, 1963) have been supplemented, if not entirely supplanted, by a wide range of disparate approaches (reviews in Szreter, 1993; Mason, 1997; Jones et al., 1998; Borgerhoff Mulder, 1998; Friedlander et al., 1999). It seems a fair appraisal that no single hypothesis is generally accepted or even largely favored as a widely applicable model for fertility decline. The increasingly rich empirical evidence, especially from microlevel studies, has led some to conclude that the causes of fertility decline are so varied and so often peculiar to specific local or regional contexts that the search for a single explanatory framework is futile (e.g., Greenhalgh, 1990; Friedlander et al., 1999). This may be overly pessimistic, especially because we have not

yet exhausted theoretical explanations that may accommodate such diversity. The call for such theory is heard from demographers (Mason, 1997; Caldwell, 1997; Hirschman, 1994) and behavioral ecologists (Bock [this volume]; Kaplan, 1996).

Economic analyses of fertility are of special interest because they share assumptions with behavioral ecology, a method based in formal modeling, and an explicit concern with the utility of children (see Robinson, 1997). This approach began with Leibenstein's (1957) attempt to formally model the process of fertility decline known as "the turning point." Leibenstein argued that fertility decreased because the utility of child services to their parents declined. Becker (1960) set these trade-offs within household economic processes, arguing that the demand for children is based on the cost efficiency by which they—rather than some other source—could produce utility for their parents. Willis (1974, 1987) noted that expanding economic roles for caretakers (primarily women) raised the opportunity cost of their time spent raising children, damping demand for offspring. Subsequently, Caldwell (1982) proposed that the turning point occurred when the net flow of wealth between generations reversed, so that children were more heavily subsidized by parents. While influential, economic approaches have been criticized on varied grounds (Robinson, 1997; Mason, 1997).

Recent applications of evolutionary theory to fertility (Kaplan 1994, 1996; Kaplan and Lancaster, 2000; Kaplan et al., 2000) extend economic models by linking them to biological approaches (see Bock, this volume). The modern demographic transition has engaged biological anthropologists and behavioral ecologists in part because the apparent association of declining fertility with increasing wealth or potential to support offspring appears to be a direct challenge to the neo-Darwinian premise that behavior is designed to promote reproductive success (e.g., Vining, 1986). Addressing this conundrum (Borgerhoff Mulder, 1998; Hill and Kaplan, 1999) has led to an emphasis on integrating life history and parental investment theory from evolutionary ecology, and human capital and other theory from economics. For instance, Kaplan's (1996) model is concerned with how people allocate resources (income) between investment in maintenance (and therefore future

reproduction) and current reproduction. What both the economic approaches and their evolutionary cousins have not done, however, is to incorporate unpredictability into fertility analyses, an effort we seek to promote in this paper.

#### *Role of mortality and unpredictability in demographic transition theory*

Claims for a role of mortality in demographic transitions date to classic formulations (e.g., Notestein, 1945). Yet, in her recent (1997) presidential address to the Population Association of American, Mason identified neglect of mortality as a flaw in many current transition theories. Concern with mortality is implicit in Easterlin's (1978, Easterlin and Crimmins, 1985) synthesis framework, which incorporates a "supply of children" component along with microeconomic and sociocultural variables, and it is retained in some other recent formulations (e.g., Cleland, 1993).

There is also explicit interest in the complex and mutual relationship between fertility and mortality, especially infant mortality, and the potential role of that relationship in the demographic transition (Montgomery and Cohen, 1998). The predominant focus is on replacement, including both physiological consequences such as earlier return to fecundity following truncation of breast feeding, and volitional or purposeful adjustment (Palloni and Rafalimanana, 1999). However, nearly all published analysis concerns the expected *level* of mortality, not variance, and it therefore addresses risk in a very general, qualitative manner, if at all. In fact, the replacement effect (or reproductive compensation) in the population literature and the variance compensation effect that we hypothesize are complementary alternatives: the more that replacement is possible, the less that general variance compensation is necessary. This is because replacement during the years of family building will reduce unpredictable variance in the number of surviving offspring.

Unpredictability in family building has been incorporated into some economic and sociological models of fertility. The relationship between child mortality and fertility is central to these (Lloyd and Ivanov, 1988). Economic models generally hold that fertility is the result of conscious decisions

based on evaluation of available options (Robinson, 1997). Higher levels of mortality are associated with lower predictability of outcome (McNicoll, 1986). Thus, Lloyd and Ivanov (1988) argue that high mortality regimes are associated with "family building by fate," which is replaced by "family building by design" in lower mortality contexts. In high-mortality populations, families adopt an "insurance strategy," producing extra births to guard against the possibility of too many of their children dying. In less unpredictable environments, with lower mortality and with greater control over fertility, insurance strategies are supplanted by "replacement strategies." In addition, where alternative means of obtaining some of the utilities of children are available (e.g., hired labor, institutional social security), adults may rely on these to deal with unpredictability in family building (Robinson, 1997). McNicoll (1986) and Lloyd and Ivanov (1988) argue that, as mortality decreases, the relationship between number of births and number of surviving offspring becomes sufficiently predictable to permit realistic reproductive planning by individual families. In contrast to this literature, we would argue that high unpredictability also is associated with planning; there are rational, adaptive responses to stochasticity as well as to predictable features of the environment. Fate, too, has its evolutionary response.

Cain (1983) explicitly argues for the potential importance of the probability of child survival and of child "default"—failure to uphold an expected role in old age support—in shaping fertility decisions. He adopts a "safety-first" approach from the agricultural development literature, and points out conditions that may lead to parents overshooting their goals in order to be on the safe side (see also Lloyd and Ivanov 1988). Cain suggests that people may be influenced more by the extremes of the distribution of experience (especially the high mortality tail) than by average experience. He thus recognizes that unpredictable variance in outcome may affect family-level fertility decisions. Based on the safety-first approach, Cain (1983) briefly proposes a two-stage fertility decline model for developing countries. Lessened mortality first reduces the excess fertility required to minimize the likelihood of low, below-target fertility outcomes (this would be positive

variance compensation in our terminology). Then, lessened hazards in old age and alternative insurance possibilities, an improved "environment of risk," weakens the need for a target fertility. Cain shows how safety-first predictions deviate from those of other models. In qualitative form, his analysis anticipates some of the results we derive from the present risk-sensitive model (see below).

#### *What is different about a risk-sensitive approach?*

Our treatment of unpredictability differs from past approaches in several ways. First, unlike the heterogeneity literature, we are concerned primarily with the behavioral consequences of unpredictable variance in fertility outcome, not with the causes of that variance. Second, we focus on odds across a range of outcomes (Table 1), rather than on the average rate of child survival. Thus, the model enables us to separate the effects of stochasticity from those of average mortality, showing that the unpredictable nature of mortality is likely to affect fertility behavior apart from average losses. Third, the existing literature that does deal with child mortality and family building focuses overwhelmingly on the negative consequences of having too few children (Lloyd and Ivanov, 1988; Cain 1983). This tends to associate unpredictability with higher fertility (to compensate for deaths or to hedge against the possibility of deaths, as in the replacement and insurance models, respectively). Our more general approach shows that unpredictability may entail either higher (*positive variance compensation*) or lower (*negative variance compensation*) fertility, depending on specifiable circumstances.

#### *Variance compensation and demographic transitions*

A full explanation of demographic transitions is not proposed, much less an alternative to extant theories. Nonetheless, a phenomenon that is generally relevant to explanation of fertility transitions has been identified and modeled. The model is applied by specifying factors that can alter either the outcome distribution or the value function so that variance compensation acts counter to high fertility. Beginning from a right-ibis value function, we consider four

classes of change that would produce a decline in predicted fertility. Each entails variance compensation, either alone or in combination with processes like those hypothesized in family economics approaches to fertility.

(1) Reduced stochastic outcome variance. Lower mortality or more predictable mortality, perhaps traced to better diet or public health measures, would lessen stochastic variance in family size outcomes and thus reduce positive variance compensation. Decline in mortality has probably been a precondition for fertility decline, despite reports of European cases in which decline in infant mortality lagged reduction in fertility (Chesnais, 1992; Mason, 1997). Given the circumstances depicted in Figure 1b, the variance compensation effect decreases from approximately three extra births to only two as survivorship increases from  $p = 0.6$  to 0.8. Thus, the VCH alone might explain why fertility levels decreased during the European demographic transition more than was required to offset the average increases in survivorship (Luttbeg et al., 2000).

(2) Elevation of the low-fertility portion of the value function. (Fig. 2a). As the value function becomes more symmetrical the stochastic and deterministic optima converge. Any change that lessens family dependence on a threshold (minimum) number of grown offspring would elevate the value of low-fertility outcomes and thereby reduce positive variance compensation. Examples might include changes in bride-price customs, provision of more secure or less expensive forms of old age subsistence insurance, or more secure property rights. The VCH will produce this effect even when the peak of the value function (the deterministic optimum) is unchanged.

(3) A sharper decline in the high-fertility portion of the value function. (Fig. 2b). A wide range of socioeconomic changes that increase the relative cost of large numbers of progeny might have this effect. For example, developing socioeconomic conditions might force or lead women into the workplace, increasing the opportunity costs of spending time raising extra children (e.g.,

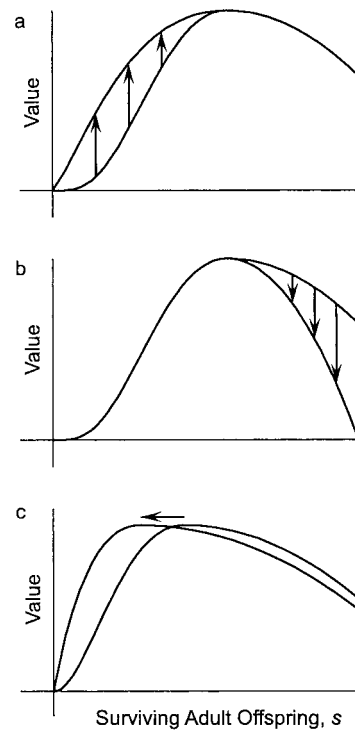


Fig. 2. Three types of change affecting a value function that can reduce positive variance compensation and consequently lessen a couple's best-choice, risk-sensitive fertility: (a) decreased costs of low fertility; (b) increased costs of higher fertility; (c) shift of peak of value function toward lower fertility.

Schultz [1985] on Sweden 1860–1910). Increasing population density or filling of the frontier may lead to difficulty in providing children with adequate land, as Easterlin (1976) and Schapiro (1982) argue was the case in the northern United States in the 19th century. Other factors that might reduce the value of higher numbers of offspring include: a shift to a household economic base less forgiving of extra children (e.g., Borgerhoff Mulder, 1987); restrictions on industrial or other employment of children; increasing difficulty in maintaining middle class socioeconomic status or increasing expectations for middle class standard of living (Banks, 1954); and higher educational costs for children.

(4) A left shift in the peak of the value curve. (Fig. 2c). A leftward shift in a right-skewed curve will mimic process (2), because it

eliminates the sharp cost of low fertility outcomes (except, of course, for  $n = 0$ ). In this case, however, the fertility decrease that is due to reduced variance compensation will be augmented by the shift in the peak of the value function.

Taking the right-ibis value function as a starting point, reduced fertility would be predicted from any socioeconomic or environmental factors that: (1) reduce the degree of unpredictable variance associated with the long-term outcome of fertility behavior early in the family cycle; (2) decrease costs of low fertility; (3) increase the costs of high fertility; or (4) displace the peak of the value function to the left. Any of these four possibilities might act in isolation, in combination, or sequentially. For instance, Cain's (1983) two-stage demographic transition model (summarized above) is equivalent to shift (1) followed by (2). The multiplicity of means by which socioeconomic and environmental variables can act through this model to affect fertility is consistent with empirical experience, showing that demographic transitions may have diverse sources of local causation. Given the range of factors that might have effects such as those cited here, risk-sensitive variance compensation may be implicated in virtually any of the existing socioeconomic hypotheses about fertility transitions.

#### *Empirical evidence of variance compensation*

Broad claims for the applicability of the VCH have been made, largely on theoretical grounds. It remains to determine empirically the magnitude of such effects. Of this we know little. The requisite data are scarce and gathering them entails methodological and conceptual hurdles. Chief among them is the problem of phenotypic correlation: empirical derivation of a value function must control for the possibility that factors native to families or their circumstances confound the relationship between completed family size and value (see Winterhalder and Smith, 2000).

Nevertheless, there is evidence that ideal family size is influenced by uncertainty in child survival. Saksena and Srivastava (1984) found that mothers in urban India considered the ideal number of children to be 2.8 if they could be certain that all would

survive, but 3.7 if survival were uncertain. Unfortunately, without knowing their expectation for average mortality ( $p$ ) under the "uncertain" scenario, we cannot use this disparity to estimate the possible magnitude of variance compensation. On the other hand, Pebley et al. (1979) found that Guatemalan women's desire for additional births was not affected by their perception of child survival, but was affected by the proportion of the women's siblings who had died in infancy or childhood. Such studies are loosely consistent with the VCH, but probably say more about the need for better understanding of how perceptions of risk are shaped and acted upon. Historical demography (Volland, 2000) and long-term ethnographic study (e.g., Borgerhoff Mulder, 1988; Hill and Hurtado, 1996) will likely play a crucial role in clarifying the relationship between risk and fertility behavior.

#### *Assumptions and potential complications*

The VCH entails two classes of assumptions: those concerning behavioral ecology, bearing mainly on the theoretical plausibility of the model (see Winterhalder and Leslie, 2002), and those pertinent to the ubiquity or magnitude of the effect, which is a largely analytic matter. Because some of the latter class of assumptions are clearly unrealistic, as befits a simple model, it is worthwhile making them and the direction of their effect explicit.

Contingent behavior and continuous adjustment. The VCH model assumes that the decision of how many children to bear is made at the beginning of a couple's reproductive career and is not changed thereafter. Stochastic events then take their toll and the outcome is experienced at the end of the family cycle. This two-stage approach is unrealistic to the degree that fertility reflects contingent, on-going adjustment, conscious or unconscious, through *stopping behavior* or *reproductive compensation* (Das Gupta, 1990; Palloni and Rafalimanana, 1999). There may be other means of hedging risk than by acquiring "extra" children (e.g., patron-client relationships; see Rosenzweig, 1988); fostering and adoption fall into the same category. However, such institutions may not satisfy all of the various values of children, especially fitness.

To the extent that these behaviors function effectively to offset stochastic elements in family building, they reduce the need to use fertility behavior to achieve risk-sensitive adaptive goals. However, the potential for continuous adjustment to reduce risk in family building is limited. Replacement of children lost demographically or socially becomes increasingly difficult as the end of the reproductive span is approached. Replacements may themselves be lost. Factors such as delayed marriage, as was characteristic of many historical European populations, limit the potential for reproductive compensation by shortening the reproductive span.

In general, possibilities for continuous adjustment through the period of family building reveal that our simplified, two-stage model of fertility choices will *overestimate* the potential importance of variance compensation.

“Mortality” by other means. We have modeled risk as arising exclusively from mortality, but other factors can contribute to unpredictability in family building and achieved value. A child may live but fail to deliver some or all anticipated welfare services, because of disability or default on expected obligations. This increases the unpredictable outcome variance associated with a given number of births, even apart from mortality.

Heterogeneity of risk. Our initial model assumes uniform risk among families, but deaths are often highly clustered (Das Gupta, 1990; Ronsmans, 1995). The same may be true of default on expected obligations. Clustering of mortality among subpopulations can arise from shared physical or biotic environment (pathogens, nutrition, etc.), cultural environment (beliefs and practices, social structure), family environment (parental competence), or genetic characteristics. By our terminology, this literature refers to heterogeneity of *hazard*, not risk. To the degree that such hazards are predictable, they are not of central concern to risk-sensitive adaptation.

However, unpredictable outcome variance (risk in our sense) may also differ among families. If the causes of mortality clustering are not apparent to those in the

community, or if the causes can be recognized only after the fact (that is, cannot be predicted), this would add to the perception of *unpredictability* and of the need to compensate for it. Change in mortality conditions over time also introduces heterogeneity between families (aggregated over time), which may or may not be perceived by the population as being associated with age or cohort differences among women. Sex differences in mortality also would increase heterogeneity, as might heterogeneity in fecundity, as this may result in shorter birth intervals and higher risk for more fecund women. Simulations show that introducing within- or between-family heterogeneity increases variance in the number of child deaths (over that in the homogeneous case, with binomial variance) (Ronsmans, 1995). Likewise, it can be shown formally that the variance of a mixed binomial—which would be required for instance if the family outcome objective depended jointly on mortality and the sex composition of the sibling set—is greater than that of either component (J. Weiss, personal communication). These observations suggest that the assumption of uniformity is a conservative one with regard to the magnitude of risk and the potential importance of variance compensation.

“Compounding” of risk. The model also assumes that the value of all offspring within a sibship is equal, and the effects of completed family size are shared equally among them. But sons and daughters may satisfy different aspects of value, in different manners and to different degrees. Furthermore, sex difference in value may itself depend on birth order (e.g., number of older brothers, as in the Turkana example given earlier). Males and females may perceive or experience the costs and benefits of children differently, generating gender-specific value functions. Position in a kinship system or socioeconomic context also may affect these evaluations. Taking account of any of these factors is likely to increase the overall unpredictability of outcome, enhancing the potential importance of variance compensation.

In general, if outcome variability arising from factors other than mortality is important, or if there is heterogeneity or compound forms of risk, then the simplified,

mortality-based model will *underestimate* the potential importance of variance compensation.

### CONCLUSIONS

"If the triumph of 19th century science was to show that nature is better viewed in terms of processes than of types, then that of the 20th c. has been to show repeatedly that the laws of nature are inextricably the laws of chance. The boundary between 'signal' and 'noise' has been forced to recede to such a degree that there is now only a very tenuous, and philosophically vulnerable, justification for separating observation into those categories." (Weiss 1976; p. 352)

One goal of this article is to bring the perspective identified by Weiss to the analysis of human fertility. In demography, stochastic effects typically are treated as noise, obscuring the "real" signals of interest. Analysts make valiant efforts, through careful attention to sample sizes, sampling design, and choice of statistical technique, to see *through* this noise, or to relegate its effects to a nonproblematic error term. But for the individuals or families that experience the actual events that comprise our statistics, deviations from statistical expectations are quite real. Unpredictable variability is fundamental to the adaptive forces that shape behavior. There are sound theoretical reasons for incorporating unpredictable fertility outcomes into analyses of reproductive strategy. This model suggests that if the value function is nonlinear, variance compensation will affect optimal reproductive strategies, and will do so apart from compensation for average level of mortality.

Good models function to highlight new empirical possibilities. In particular, the VCH points to the need for data useful in estimating or deriving outcome distributions and value functions. Microlevel data, especially carefully collected individual reproductive histories, have the potential to reveal much about outcome distributions. Such data exist, but need to be linked to suitable value functions. When the metric of interest is reproductive success (fitness), reproductive histories themselves, especially if expanded over two or more generations, will provide part of the information needed.

Given the current availability of data on ideal family size, parity, and number of living children, a focus on completed family size, as we have taken here, makes sense. Future applications may find that a shift in focus is fruitful or even necessary. For example, decision making about reproduction in many cases is likely to be directed at spacing and timing of births, which have clear consequences for maternal and child health, rather than target number of grown children. Costs of children to some extent depend on the spacing of births and the age and availability of siblings; a focus on ideal and achieved family size and composition, characteristic of much demographic research, may be misleading. However, variance compensation should retain its relevance as the focus shifts among different fertility-related outcomes.

Variance compensation can influence reproductive behavior in ways that may significantly increase or decrease fertility relative to expectations derived from deterministic models. Here, we have emphasized the relevance of risk-sensitive adaptive tactics for understanding demographic transitions, but other applications are possible (Winterhalder and Leslie, 2002; Winterhalder et al., manuscript in preparation). Through its effect on fertility behavior, variance compensation has a direct bearing on birth spacing and completed fertility, and thereby on problems in demography and human population biology ranging from fertility patterns to maternal depletion and child health. Others in this symposium have pointed to the great potential of evolutionary theory as a basis for articulating a general theory of fertility and human reproduction. Variance compensation will be an element of such theory.

### ACKNOWLEDGMENTS

Jack Weiss developed the mathematical expression of the value function used to illustrate the model and to generate Figures 1 and 2. For encouragement and insightful, constructive criticism we thank: Monique Borgerhoff Mulder (and the students of the behavioral ecology lab, UC Davis), Sheryl Gerety, Eric Alden Smith, Barbara Entwisle, Hillard Kaplan, Katherine Wildman, and various participants in the Carolina Population Center, Friday Seminar.

## LITERATURE CITED

- Banks JA. 1954. Prosperity and parenthood. London: Routledge and Kegan Paul.
- Becker G. 1960. An economic analysis of fertility. In: Demographic and economic change in developed countries. NBER Series 11. Princeton, NJ: National Bureau of Economic Research.
- Blurton Jones NG, Sibly RM. 1978. Testing adaptiveness of culturally determined behavior: Do bushman women maximize their reproductive success by spacing births widely and foraging seldom? In: Blurton Jones NG, Reynolds V, editors. Human behavior and adaptation. London: Taylor and Francis. p 135-157.
- Blurton Jones NG. 1987. Bushman birth spacing: A direct test of simple predictions. *Ethol Sociobiol* 8:183-203.
- Bock J. 1995. Determinants of variation in children's activities in a southern African community. Doctoral dissertation, Dept. of Anthropology, University of New Mexico.
- Bock J. (in press) (ms. in this volume).
- Bongaarts J. 1984. Building a family: Unplanned events. *Stud Fam Plan* 15:14-19.
- Boone JL, Kessler KL. 1999. More status or more children? Social status, fertility reduction and long-term fitness. *Evol Hum Behav* 20:257-277.
- Borgerhoff Mulder M. 1987. Resources and reproductive success in women with an example from the Kipsigis of Kenya. *J Zool (London)* 213:489-505.
- Borgerhoff Mulder M. 1988. Reproductive success in three Kipsigis cohorts. In: Clutton-Brock TH, editor. Reproductive success. Chicago: University of Chicago Press. p 419-435.
- Borgerhoff Mulder M. 1992. Demography of pastoralists: Preliminary data on the Datoga of Tanzania. *Hum Ecol* 20:383-405.
- Borgerhoff Mulder M. 1998. Demographic transition: Are we any closer to an evolutionary explanation? *Trends Ecol Evol* 13:266-270.
- Cain M. 1977. The economic activities of children in a village in Bangladesh. *Popul Devel Rev* 3:207-221.
- Cain M. 1983. Fertility as an adjustment to risk. *Popul Devel Rev* 9:688-702.
- Caldwell JC. 1982. Theory of fertility decline. London: Academic Press.
- Caldwell JC. 1997. The global fertility transition: The need for a unifying theory. *Popul Devel Rev* 23: 803-812.
- Chesnais J-C. 1992. The demographic transition: stages, patterns, and economic implications. A longitudinal study of sixty-seven countries covering the period 1720-1984. Translated by Kreager E, Kreager P. Oxford: Clarendon Press.
- Cleland J. 1993. Equity, security and fertility: A reaction to Thomas. *Popul Stud* 47:345-352.
- Curtis SL, Diamond I, McDonald JW. 1993. Birth interval and family effects on postneonatal mortality in Brazil. *Demography* 30:33-43.
- Das Gupta M. 1990. Death clustering, mothers' education and the determinants of child mortality in rural Punjab, India. *Popul Stud* 44:489-505.
- Davis K. 1945. The world demographic transition. *Ann Am Acad Polit Soc Sci* 237:1-11.
- Davis K. 1963. The theory of change and response in modern demographic history. *Popul Index* 29:345-366.
- Easterlin RA. 1976. Population change and farm settlement in the northern United States. *J Eco Hist* 36:45-75.
- Easterlin RA. 1978. The economics and sociology of fertility: a synthesis. In: Tilly C, editor. Historical studies in changing fertility. Princeton: Princeton University Press. p 57-133.
- Easterlin RA, Crimmins E. 1985. The fertility revolution: A supply-demand analysis. Chicago: University of Chicago Press.
- Elster J. 1986. Introduction. In: Elster J, editor. Rational choice. New York: New York University Press. p 1-33.
- Foley R. 1985. Optimality theory in anthropology. *Man (N.S.)* 20:222-242.
- Friedlander D, Okun BS, Segal S. 1999. The demographic transition then and now: Processes, perspectives, and analyses. *J Fam Hist* 24:493-533.
- Friedman D, Hechter M, Kanazawa S. 1994. A theory of the value of children. *Demography* 31:375-401.
- Gardiner E, Yerushalmy J. 1939. Familial susceptibility to still births and neo-natal deaths. *Am J Hyg* 30:11-31.
- Greenhalgh S. 1990. Toward a political economy of fertility: anthropological contributions. *Popul Dev Rev* 16:85-106.
- Hill K, Hurtado M. 1996. Ache life history. The ecology and demography of a foraging people. New York: Aldine de Gruyter.
- Hill K, Kaplan H. 1999. Life history traits in humans: Theory and empirical studies. *Annu Rev Anthropol* 28:397-430.
- Hirschman C. 1994. Why fertility changes. *Annu Rev Socio* 20:203-233.
- Hobcraft JN, McDonald JW, Rutstein SO. 1985. Demographic determinants of infant and early childhood mortality: A comparative analysis. *Popul Stud* 39:363-385.
- Hull TH. 1995. The value of children at the advent of fertility decline in Java. *J Popul* 1:175-202.
- Huttly SR, Victora CG, Barros FC, Vaughan JP. 1992. Birth spacing and child health in urban Brazilian children. *Pediatrics* 89:1049-1054.
- Jones GW, Douglas RM, Caldwell JC, D'Souza RM, editors. 1998. The continuing demographic transition. New York: Oxford University Press.
- Kaplan H. 1994. Evolutionary and wealth flows theories of fertility: Empirical tests and new models. *Popul Dev Rev* 20:752-791.
- Kaplan H. 1996. A theory of fertility and parental investment in traditional and modern human societies. *Yrbk Phys Anthropol* 39:91-135.
- Kaplan H, Hill K, Lancaster J, Hurtado AM. 2000. A theory of human life history evolution: diet, intelligence and longevity. *Evol Anthropol* 9:156-185.
- Kaplan H, Lancaster JB. 2000. The evolutionary economics and psychology of the demographic transition to low fertility. In: Cronk L, Chagnon NA, Irons W, editors. Human behavior and adaptation: An anthropological perspective. New York: Aldine de Gruyter p 283-323.
- Lee RD, Bulatao RA. 1983. The demand for children: A critical essay. In: Bulatao RA, Lee RD, editors. Determinants of fertility in developing countries. Vol. 1. Supply and demand for children. New York: Academic Press. p 233-287.
- Leibenstein HM. 1957. Economic backwardness and economic growth. New York: John Wiley.
- Leslie PW, Dyson-Hudson R. 1999. People and herds. In: Little MA, Leslie PW, editors. Turkana herders of the dry savanna. Ecology and biobehavioral response of nomads to an uncertain environment. Oxford: Oxford University Press. p 233-247.
- Leslie PW, Dyson-Hudson R, Fry PH. 1999. Population replacement and persistence. In: Little MA, Leslie PW, editors. Turkana herders of the dry savanna. Ecology and biobehavioral response of nomads to an

- uncertain environment. Oxford: Oxford University Press. p 281–301.
- Little MA, Leslie PW, Campbell KL. 1992. Energy reserves and parity of nomadic and settled Turkana women. *Am J Hum Biol* 4:729–738.
- Lloyd CD, Ivanov S. 1988. The effects of improved child survival on family planning practice and fertility. *Stud Fam Plan* 19:141–161.
- Low BS, Clarke AL, Lockridge KA. 1992. Toward an ecological demography. *Popul Dev Rev* 18:1–31.
- Luttbeg B, Borgerhoff Mulder M, Mangel M. 2000. To marry again or not? A dynamic model for demographic transition. In: Cronk L, Chagnon NA, Irons W, editors. *Human behavior and adaptation: An anthropological perspective*. New York: Aldine de Gruyter p 345–368.
- Mason KO. 1997. Explaining fertility transitions. *Demography* 34:443–454.
- McNicol G. 1986. Adaptation of social systems to changing mortality regimes. In: *Consequences of mortality trends and differentials*. Dept of International Economic and Social Affairs, United Nations, Population Studies, No. 95. p 13–19.
- Montgomery M, Cohen B, editors. 1998. *From death to birth: Mortality decline and reproductive change*. Washington, DC: National Academy press.
- Notestein F. 1945. Population—the long view. In: Schultz TW, editor. *Food for the world*. Chicago: University of Chicago Press. p 36–57.
- Palloni A, Rafalimanana H. 1999. The effects of infant mortality on fertility revisited: New evidence from Latin America. *Demography* 36:41–58.
- Pebley AR, Delgado H, Brinemann E. 1979. Fertility desires and child mortality experience among Guatemalan women. *Stud Fam Plan* 10:129–136.
- Randall S. 1984. *The demography of three Sahelian populations: Marriage and childcare as intermediate determinants of fertility and mortality*. Doctoral dissertation, London University.
- Robinson WC. 1997. The economic theory of fertility over three decades. *Popul Stud* 51:63–74.
- Ronsmans C. 1995. Patterns of clustering of child mortality in a rural area of Senegal. *Popul Stud* 49:443–461.
- Rosenzweig M. 1988. Risk, implicit contracts and the family in rural areas of low-income countries. *Econ J* 98:1148–1170.
- Saksena DN, Srivastava JN. 1984. Impact of child mortality and sociodemographic attributes on family size desires: Some data from urban India. *J Biosoc Sci* 16:119–126.
- Schapiro MO. 1982. Land availability and fertility in the United States, 1760–1870. *J Eco Hist* 42:577–600.
- Schultz TP. 1985. Changing world prices, women's wages, and the fertility transition: Sweden, 1860–1910. *J Polit Econ* 93:1126–1154.
- Smith EA, Winterhalder B. 1992. Natural selection and decision-making: Some fundamental principles. In: Smith EA, Winterhalder B, editors. *Evolutionary ecology and human behavior*. New York: Aldine de Gruyter. p 25–60.
- Stash S. 1996. Ideal-family-size and sex-composition preferences among wives and husbands in Nepal. *Stud Fam Plan* 27:107–118.
- Stevens DW, Charnov EL. 1982. Optimal foraging: some simple stochastic models. *Behav Ecol* 10:251–263.
- Szreter S. 1993. The idea of demographic transition and the study of fertility change: A critical intellectual history. *Popul Dev Rev* 19:659–701.
- Thompson WS. 1929. Population. *Am Sociol Rev* 34:959–975.
- Toulmin C. 1992. *Cattle, women, and wells. Managing household survival in the Sahel*. Oxford: Clarendon Press.
- Tracer DP. 1991. Fertility-related changes in maternal body composition among the Au of Papua New Guinea. *Am J Phys Anthropol* 85:393–405.
- Trussell J, Rodriguez G. 1990. Heterogeneity in demographic research. In: Adams J, Lam D, Hermalin A, Smouse P, editors. *Convergent issues in genetics and demography*. London: Oxford University Press. p 111–132.
- Vining DR. 1986. Social versus reproductive success: the central theoretical problem of human sociobiology. *Behav Brain Sci* 9:167–216.
- Voland E. 1995. Reproductive decisions viewed from an evolutionarily informed historical demography. In: Dunbar RIM, editor. *Human reproductive decisions*. New York: St. Martin's Press. p 137–159.
- Voland E. 2000. Contributions of family reconstitution studies to evolutionary reproductive ecology. *Evol Anthropol* 9:134–146.
- Weiss KM. 1976. Demographic theory and anthropological inference. *Ann Rev Anthropol* 5:351–81.
- Winterhalder B, Smith EA. 1992. Evolutionary ecology and the social sciences. In: Smith EA, Winterhalder B, editors. *Evolutionary ecology and human behavior*. New York: Aldine de Gruyter. p 3–23.
- Winterhalder B, Lu F, Tucker B. 1999. Risk-sensitive adaptive tactics: Models and evidence from subsistence studies in biology and anthropology. *J Archaeol Res* 7:301–348.
- Winterhalder B, Smith EA. 2000. Analyzing adaptive strategies: Human behavioral ecology at twenty-five. *Evol Anthropol* 9:51–72.
- Winterhalder B, Leslie P. 2002. Risk-sensitive fertility: the variance compensation hypothesis. *Evol Human Behav* 23:59–82.
- Willis RJ. 1974. Economic theory of fertility behavior. In: Schultz TW, editor. *Economics of the family: Marriage, children, and human capital*. Chicago: University of Chicago Press. p 25–75.
- Willis RJ. 1987. What have we learned from the economics of the family? *Am Econ Rev* 77:68–81.
- Wood JW. 1994. *Dynamics of human reproduction. Biology, biometry, demography*. New York: Aldine de Gruyter.
- Zaba B, David P. 1996. Fertility and the distribution of child mortality risk among women: An illustrative analysis. *Popul Stud* 50:263–278.

#### APPENDIX: CONSTRUCTING A RIGHT BIS FUNCTION

The ibis function used to produce the figures in this article is a three-parameter piecewise-defined function constructed by linking a member of the family  $f(x) = x^a e^{-bx}$  to a member of the family of quadratic polynomials. The function's parameters specify the location of the local maximum, inflection point, and nonzero root. The general formula is

$$I(x; m, n, r) = \begin{cases} 0, & \text{if } x < 0 \\ e^{\frac{-m^2}{(m-n)^2} - \frac{mx}{(m-n)^2} - \frac{m^2}{x(m-n)^2} - \frac{m^2}{(m-n)^2}}, & \text{if } 0 \leq x < m \\ -\frac{(x-m)^2}{(m-r)^2} + 1, & \text{if } m \leq x < r \\ 0, & \text{if } x \geq r \end{cases}$$

where  $m$  is the location of the local maximum,  $n$  is the location of the inflection point, and  $r$  is the location of the nonzero root. The parameter values used for Figure 1 are  $m = 4$ ,  $n = 2$ ,  $r = 18$ .