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Resource Availability, Mortality, and Fertility: A Path Analytic Approach to Global Life-History Variation

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Abstract Humans exhibit considerable diversity in timing and rate of reproduction. Life-history theory (LHT) suggests that ecological cues of resource richness and survival probabilities shape human phenotypes across populations. Populations experiencing high extrinsic mortality due to uncertainty in resources should exhibit faster life histories. Here we use a path analytic (PA) approach informed by LHT to model the multiple pathways between resources, mortality rates, and reproductive behavior in 191 countries. Resources that account for the most variance in population mortality rates are predicted to explain the most variance in total fertility rates. Results indicate that resources (e.g., calories, sanitation, education, and health-care expenditures) influence fertility rates in paths through communicable and noncommunicable diseases. Paths acting through communicable disease are more strongly associated with fertility than are paths through noncommunicable diseases. These results suggest that a PA approach may help disaggregate extrinsic and intrinsic mortality factors in cross-cultural analyses. Such knowledge may be useful in developing targeted policies to decrease teenage pregnancy, total fertility rates, and thus issues associated with overpopulation.

Human populations exhibit considerable variation in timing and frequency of reproduction. Adolescent fertility rates in Niger, for example, were over 31 times higher than in South Korea (World Health Organization 2009). Variation in fertility rates across populations result, in part, from differences in sources of mortality (Roff 2002; Stearns 1992). Sources of mortality may respond differently to social resources (e.g., access to health care, water, and sanitation services; education; income equality, etc.). Here we test hypotheses from LHT concerning the nature of mortality (i.e., intrinsic vs. extrinsic) and mortality effects on reproduction. In general, LHT predicts that high mortality rates cue fast life histories which are characterized by early reproduction and relatively low parental investment per offspring (Borgerhoff Mulder 1992; Bulled and

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Sosis 2010; Chisholm 1993; Low, Hazel, Parker, and Welch 2008; Nettle 2010; Promislow and Harvey 1990; Quinlan 2010; Roff 2002; Stearns 1992). A key feature of LHT, however, divides mortality causes into intrinsic and extrinsic components. Those components of mortality have proved to be exceedingly difficult to isolate empirically. Here we use a PA approach to untangle relations between resources, disease, mortality, and fertility. This approach allows us to begin to assess effects of extrinsic and intrinsic components of mortality (and the resources associated with each) on human reproductive behavior.

Life-History Theory. LHT provides an evolutionary framework for understanding how environmental cues of resource richness and organismal survivorship affect reproductive decisions. Theoretically, the evolution and development of life-history strategies trend toward enhancing individual reproductive fitness in specific environments (Roff 2002; Stearns 1992). Adaptive life-history strategies develop, in part, in response to costs and benefits of allocating energy (i.e., resources) to growth, maintenance, and reproduction within variable ecological contexts. As resources invested into one life function (e.g., mating) cannot be devoted to another (e.g., growth), trade-offs arise (Stearns 1989).

One of the most fundamental trade-offs in an organism's life-history is between current versus future reproduction (see review in Ellis, Figueredo, Brumbach, and Schlomer 2009). Fitness costs and benefits associated with this trade-off are guided by variation in life-expectancy and quality versus quantity of offspring. Delaying reproduction allows an organism to allocate more resources to somatic effort (i.e., growth and maintenance), thereby lengthening life-expectancy and increasing the ability to produce and invest in higher quality offspring. This delay, by decreasing energy devoted to reproductive effort, lowers the quantity of offspring across the reproductive life-span. In contrast, earlier investment in reproduction increases the quantity of potential offspring across the reproductive life-span but shortens remaining life-expectancy as maternal somatic resources are depleted through repeated pregnancy and lactation. Earlier and more frequent investment in reproduction, by limiting somatic investment, decreases the quality of offspring by reducing the amount of parental investment per offspring (Roff 2002; Stearns 1992). A potentially complex relationship exists between the allocation of energy to somatic and reproductive effort and life-expectancy. Over the life-course, increased investment in somatic effort should lengthen life-span relative to other allocation decisions within a population. However, a longitudinal study of mortality and reproduction indicated that population mortality rates in early life had a causal role in the allocation of both somatic and reproductive effort later in adulthood, when early life and later population mortality rates were uncorrelated (Quinlan 2010).

Environmental Risk and Life-History. In LHT, the local nature of risk is a major factor affecting trade-offs in the allocation of effort to somatic maintenance, development, mating, and parenting (Chisholm 1999; Quinlan 2007,

2010). LHT partitions risk into two types: extrinsic and intrinsic. Extrinsic mortality is the risk of death that is not conditional on an organism's reproductive behavior (Stearns 1992:182). Statistically, we can define extrinsic mortality as variance in the probability of death that is not accounted for by mating effort or parenting effort (or by extension trade-offs between reproductive and somatic effort). In other words, an organism cannot escape extrinsic mortality by changing its behavior. It is the age-specific risk of death is equally shared by all members of a population. Intrinsic mortality, in contrast, is the probability of death associated with allocation of both somatic and reproductive effort. Predation, for example, could be either extrinsic or intrinsic mortality or both. Imagine a population of organisms in which a probability (P) of death exists from predation at age X. Then P is a combination of factors: some are beyond an individual's control but others are not. The frequency by which an individual encounters a predator depends on extrinsic factors such as the density of predators in the environment (beyond the individual's control) *and* intrinsic factors such as the level of vigilance, time spent exposed in the landscape as a result of mating effort, etc. (determined by allocation of effort). An individual of a prey species in an environment with many predators may reduce the probability of death by predation by adjusting its behavior, but always some extrinsic probability of death by predation exists. The predation example raises an important point about extrinsic mortality: Any age-specific probability of death has both intrinsic and extrinsic components that can be difficult to isolate analytically. Despite empirical challenges, extrinsic and intrinsic components of mortality can have profound influences on adaptive behavior.

Extrinsic mortality plays a key role in the evolution of life histories and reproductive strategies (Chisholm 1993, 1999; Promislow and Harvey 1990; Roff 2002; Stearns 1992). When extrinsic mortality is high, then organisms should reproduce early in life to reduce mortality exposure over time and to 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. Conversely, in high-risk environments, early reproduction and minimal parental investment per offspring can be adaptive. These predicted relationships hold among mammals: Juvenile mortality is 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).

Extrinsic risk for humans has attracted theoretical interest since the early 1990s (e.g., Borgerhoff Mulder 1992; Chisholm 1993, 1999; Harpending, Draper, and 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 and Daly 1997), sub-Saharan Africans (Gant, Heath, Ejikeme, Snell, and Briar-Lawson 2009), and in four cross-cultural studies (Bulled and Sosis 2010; Low et al. 2008; Placek and Quinlan 2011; Walker et al. 2006). Nettle (2010) documented similar relationships across British neighborhoods where economically marginalized (i.e., lower resource availability) neighborhoods displayed earlier ages at reproduction, lower birth weights, and shorter durations of breastfeeding (see also Nettle, Coall, and Dickins 2011). In a longitudinal study of a rural Dominican community, Quinlan (2010) found that high infant-mortality rates predicted earlier ages of first reproduction, although very high infant-mortality rates produced a saturation point of parental investment resulting in reproductive delays. Support for a relationship between mortality rates and life-history strategies is also documented among hunter−gatherer and small-scale horticulturalist groups (Walker et al. 2006). And even the perception of mortality may influence human-reproductive behavior (Chisholm, Quinlivan, Petersen, and Coall 2005). This small body of research makes 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? How do we empirically distinguish between extrinsic and intrinsic components of mortality? Here we use a path analytic approach to identify specific factors mediating and moderating effects on fertility rates across 191 nations.

Although the distinction between extrinsic and intrinsic mortality is critical for LHT predictions, partitioning mortality into extrinsic or intrinsic components has proved very difficult (Ellis et al. 2009). In many studies, mortality rates are commonly quantified with "all-cause" mortality parameters, such as life-expectancy at birth (LEB) or parameters exhibiting strong correlations with LEB, such as infant mortality (see Anderson 2010; Bulled and Sosis 2010; Low et al. 2008; Wilson and Daly 1997). Even studies using "all-cause" mortality measures across and within taxa have demonstrated a strong positive relationship between higher rates of mortality and faster life-history strategies. Better predictive models and theoretical development await improved analytical strategies that can identify components of mortality.

Resources and Life-History. Studies across and within human populations, in agreement with findings across numerous nonhuman species, indicate that relatively higher mortality rates are associated with both earlier onset and higher rate of reproduction (Anderson 2010; Bulled and Sosis 2010; Low et al. 2008; Promislow and Harvey 1990; Quinlan 2010; Stearns 1992). Unlike any other species, however, humans are capable of producing resources that lead to increases and decreases in survival probabilities of mortality causes and thus may play a direct role in population mortality variation. For example, access to medicinal resources can increase the survival probability of certain diseases while weapons of modern warfare can decrease the survival probability of

conflict. This capacity is important in a life-history framework as resources may transform an extrinsic cause of mortality to an intrinsic cause. For instance, malaria may be defined as a source of extrinsic risk when individuals lack access to necessary medication or preventative measures. When medicines/preventative measures become available, however, somatic investment (e.g., searching for employment in order to afford medicine) can increase the survival probability associated with malaria, thus making malaria an intrinsic cause of morality. Theoretically, we expect access to malaria medicines/preventative measures will lead to increases in a population's LEB and thus alter the influence of mortality from malaria on life-history strategies. Critically, a cause of mortality previously associated with "faster" life-history strategies now cues the development of "slower" strategies.

Previous studies examining the relationship between resources and life-history strategies in humans have primarily focused on indirect proxies of resource availability, such as participation in education/workforce, and their associations with mortality (Bulled and Sosis 2010; Low et al. 2008; Wilson and Daly 1997). Theoretically, investment in education and employment, by representing an increase in somatic investment, should coincide with delays in reproduction and increases in LEB. Further, participation in education/workforce should increase as population-mortality rates fall, thereby increasing the probability that future benefits of an education and employment will be accrued (Hill and Kaplan 1999; Kaplan, Hill, Lancaster, and Hurtado 2000). Empirical support for a relationship between investment in education/workforce and longer LEB has found some support across cultures. Low et al. (2008) documented a significant moderate-to-strong correlation between LEB and female secondary school enrollment of ($r = 0.405$, $P < 0.05$). However, female participation in the workforce did not have a significant correlation with LEB. This nonsignificant effect may arise because a large majority of females in developing countries with comparatively low LEB are employed in the agricultural sector (Low et al. 2008). Bulled and Sosis (2010) documented a similar relationship with LEB displaying a strong positive relationship with adult literacy rate ($r = 0.699$, $P < 0.01$), overall school enrollment ($r = 0.699$, $P < 0.01$), secondary school enrollment ($r = 0.810$, $P < 0.01$), and tertiary school enrollment ($r = 0.676$, $P < 0.01$). However, a significant relationship did not exist between LEB and primary school enrollment ($r = 0.103$, $P < 0.05$). The authors suggest this nonsignificant effect indicates that a threshold of educational attainment must be reached (i.e., secondary) before effects on LEB are noticeable.

Predictions. The current article examines the trade-off between current and future reproduction in 191 countries by testing two hypotheses about the onset and frequency of female reproduction. Female fertility in the 15−19 cohort is used as proxy for early reproduction. Frequency of reproduction in females is represented by total fertility rates. Differences in adolescent and total fertility rates across nations reflect variation in life-history strategies on the fast to slow

spectrum, with earlier reproduction and higher rates indicating faster life-history strategies and later reproduction and lower rates indicating slower strategies. The first hypothesis tested is that *causes of mortality with the greatest impact on population mortality rates will have the largest impact on adolescent and total fertility rates*. Causes of mortality with the greatest impact on population mortality rates include those that impact survival associated with younger age cohorts, because mortality rates in younger cohorts have a greater relative impact on LEB than in older cohorts. Mortality causes that preferentially impact younger age cohorts, especially in children under five, are often communicable diseases (e.g., HIV, malaria, pneumonia) (Leowski 1986; Lopez, Mathers, Ezzati, Jamison, and Murray 2006; Sachs and Malaney 2002). Hence, we predict that *mortality attributable to communicable diseases accounts for more variance in adolescent and total fertility rates than noncommunicable diseases*. Beyond differences in transmission, communicable diseases (e.g., malaria, tropical cluster diseases) exhibit a larger impact on younger age cohorts, especially infants, whereas many noncommunicable diseases (e.g., cancer, type 2 diabetes) have greater impacts on older cohorts. Building upon this, the second hypothesis tested is that *resources with the greatest impact on the survival probabilities of communicable diseases will have the greatest impact on adolescent and total fertility rates.* Resources affecting survival probabilities of communicable diseases are those affecting transmission environments and the availability of health care (e.g., medicine and preventative measures) (Watson, Gayer, and Connolly 2007). Based on this reasoning, we predict *resources affecting the transmission environment and the availability of treatment and preventative measures will have the largest impact on the survival probabilities associated with communicable diseases*.

Materials and Methods

Data Analysis. A PA approach was used to model the relationships between resources, mortality parameters, and total fertility rates. PA is an extension of multiple regression where regression is conducted over a set of variables. Results of a PA, called "path coefficients," reflect the magnitude and statistical significance of the predicted relationships across the set of variables. PA has a number of analytical strengths compared to the ordinary least squares (OLS) regression techniques used in previous studies (e.g., Bulled and Sosis 2010; Low et al. 2008; Wilson and Daly 1997). Most important among these is the ability to correctly specify the form and complexity of life-history relationships, more specifically the causal relationships predicted to operate between resources, morality, and life-history strategies. Enabling this specification is the use of mediator variables, which act as both dependent and independent variables. As both dependent and independent variables, mediator variables allow for the quantification of the indirect relationships, referred to as indirect effects, which are predicted to exist between a set of variables (e.g., resources, mortality, and fertility). Calculation of indirect effects allows for more nuanced tests of

Table 1. Variable Labels, Years, and Sources

Variable	*Label*	*Year(s)*	*Source*
Total fertility rate	TFR	2007	World Health Organization
Age-specific fertility 15−19 y.o.	ASF	2007	World Development Indicator
Life-expectancy at birth	LEB	2005	World Development Indicator
Years lost to communicable disease	Comm	2004	World Health Organization
Years lost to noncommunicable disease	Noncomm	2004	World Health Organization
Calories per capita	Calorie	2000−2003	World Health Organization
Total healthcare expenditure	Health	2000	World Health Organization
Female literacy rate	Femlit	2000−2001	Gender Info
GINI	GINI	1999−2003	Human Development Report
Access to clean water and sanitation	Clean	2000	World Health Organization
Contraception-prevalence rate	CPR	1999−2003	State of the World's Children

life-history predictions because the effect of resources on life-history strategies is likely mediated through a resource's prior impact on population mortality rates. For example, access to clean water, while it may not directly impact adolescent fertility rates, indirectly impacts these rates through prior direct effects on mediator variables that *do* have direct effects on adolescent fertility rates, such as population mortality rates. Indirect effects are calculated as the product of the direct effects. Both direct and indirect effects are interpreted as standardized regression coefficients.

Data Sources. Data used in the analysis was gathered from several online databases at the UN Data portal (http://data.un.org) on 191 United Nations countries. Resource variables represent data from years 1999−2003, causes of mortality variables are taken from 2004, LEB from 2005, and fertility data from 2007. It would have been preferable to use resource variables collected in the same year. However, since data for resources are not collected every year for every country, it was not possible to find a year in which all resource variables were collected. Data primarily are derived from civil registration records, and/or surveys and censuses. Variable descriptions, labels, years, and data sources are provided in Table 1.

Data Definitions. Fertility rate indicators included in the models were total fertility rate and age-specific fertility for females 15 to 19 years old. Total fertility rate (*TFR*) is the average number of births expected across a female's reproductive life-span if current age-specific fertility rates remained constant. Age-specific fertility (*ASF*) is defined as the number of births per 1,000 women in a given age range. Mortality rate indicators included in the model were life expectancy at birth (*LEB*) and years lost to communicable (*comm*) and noncommunicable diseases (*noncomm*). Years lost to communicable diseases reflect a percentage of the distribution of years of life lost to communicable disease. Years lost to noncommunicable disease are age-standardized mortality rates for noncommunicable diseases. A complete list of the diseases included in the

calculation of years lost to communicable and noncommunicable diseases can be accessed at www.who.int. Resource indicators included in the model were access to clean water and sanitation services (*clean*), total health-care expenditure (*health*), calories per capita (*calorie*), GINI (*GINI*), adult female literacy (*femlit*), and contraception prevalence rate (*CPR*). Access to clean water and sanitation is a percentage reflecting the proportion of the population using improved drinking water and sanitation facilities. Percentages of access to clean water and sanitation were combined into a composite variable reflecting the proportion of the population with access to both clean water and sanitation facilities. Total expenditure on health care reflects the per capita expenditure from both government and nongovernmental agencies on health-care services. Per capita values are in US dollars and are based off the purchasing-power parity. Years lost to communicable diseases reflect a percentage of the distribution of years of life lost to communicable disease. The GINI coefficient is an indicator of income inequality where a score of 0 indicates complete equality and 1 indicating complete inequality in income. Data on adult female literacy rates reflect females age 15 and above. Contraceptive prevalence rate includes both modern and traditional methods.

All analyses were done in M*plus* (version 6.1; Muthén and Muthén 2010) and Stata (version 11; StataCorp 2009). As a previous study by Quinlan (2010) documented a quadratic relationship between mortality and age at first birth, quadratic effects between mortality parameters (i.e., LEB and mortality causes) and fertility parameters (i.e., adolescent and total fertility) were modeled but were not significant. Likewise, the potential for interaction effects among resource variables was tested but did not result in a better fitting model. Several variables were missing data from a few countries. A benefit of M*plus* is that it uses a full-information maximum likelihood estimator which uses all available data, (i.e., N = total sample size), including cases with missing data (Brown 2006). Although the amount of missing data was small, a description of the missing data is provided in covariance coverage matrices in Appendix C. Due to significant levels of skewness and kurtosis in some variables (see Table 2), a maximum likelihood estimator with robust standard errors (MLR) was used. Correlations among the variables are provided in Table 3.

Results

Model Fit. MLR estimation converged on an admissible solution for both path models. Global and localized fit indices indicate both models displayed good overall fit (see Table 4). Model chi-squares were nonsignificant, model 1: $X^2_{\mathrm{M}} = 17.62$ ($P = 0.309$, $df_{\mathrm{M}} = 15$), model 2: $X^2_{\mathrm{M}} = 3.90$ ($P = 0.79$, $df_{\mathrm{M}} = 7$), and so the exact-fit hypothesis, (i.e., no discrepancies between population and model predicted matrix) cannot be rejected (Kline 2010). The Standardized Root Mean Square Residual (SRMR) value, which can be conceptualized as the average discrepancy between the correlations in the matrix of observed values and those in the model predicted matrix, was below the suggested 0.08 value for both

Table 2. Variable Parameters[a]

Variable	*M*	*Min−max*	*SD*	*Skew*	*Kurtosis*
TFR	2.94	1.2−7.2	1.53	1.05	3.24
ASF	53.14	3.16−201.41	43.58	0.98	3.13
LEB	66.7	41.21−82.10	10.65	−0.73	2.41
Comm	37.40	31−87	28.08	0.42	1.73
Noncomm	681.46	284−1309	200.29	0.00	2.58
Calorie	2689.78	1557−3814	505.83	0.16	2.39
Health	631.67	1−4570	879.29	2.05*	7.02*
Femlit	78.29	12.6−99.9	24.41	−1.07	2.97
GINI	40.86	24.7−74.3	9.42	0.50	2.99
Clean	74.63	18−100	23.84	−0.59	2.06
CPR	47.39	3−96	22.49	−0.13	2.00

See Table 1 for explanation of variable label. * Significant at $P < 0.05$.

models (Brown 2006). For model 2, the Root Mean Square Error of Approximation (RMSEA) and the associated 90% C.I. were below the suggested .06 cut-off criteria (Hu and Bentler 1999). For model 1, the upper level of the 90% C.I. for the RMSEA was above the suggested 0.06 cut-off criteria but still below 0.08, which is consistent with a mediocre model fit (MacCallum, Browne, and Sugawara 1996). Hu and Bentler (1999), however, note that the RMSEA test tends to over-reject models with small sample sizes, which characterizes the current sample ($n = 191$). Evaluation of model fit through comparative fit indices, which compare the model to a more restricted or "parsimonious" model, provides further evidence of good fit for both path models. Both the Comparative Fit Index (CFI) and the Tucker Lewis Index (TLI) values were above the suggested 0.95 cut-off criteria (Hu and Bentler 1999).

Localized fit indices indicated good overall fit for both path models. For model 2, inspection of modification indices (MI), which indicate the increase in model X^2, revealed no areas of localized ill-fit. Inspection of the MI for model 1 revealed no areas of ill-fit with the exception of two parameters, a direct path between *ASF* and *clean* (MI = 4.545) and a correlation between *femlit* and *LEB*

Table 3. Variable Correlations[a]

	TFR	*ASF*	*LEB*	*Comm*	*Noncomm*	*Calorie*	*Health*	*Clean*	*GINI*	*Femlit*	*CPR*
TFR	1										
ASF	0.783	1									
LEB	−0.781	−0.717	1								
Comm	0.822	0.745	−0.917	1							
Noncomm	0.578	0.450	−0.713	0.592	1						
Calorie	−0.607	−0.599	0.722	−0.733	0.623	1					
Health	−0.454	−0.466	0.602	−0.601	0.672	0.703	1				
Clean	−0.728	0.718	0.836	−0.834	−0.572	0.661	0.538	1			
GINI	0.276	0.431	−0.345	0.427	0.120	−0.361	−0.372	−0.288	1		
Femlit	−0.735	−0.634	0.690	−0.752	−0.509	0.499	0.463	0.755	−0.184	1	
CPR	−0.754	−0.510	0.697	−0.698	−0.576	0.533	0.444	0.677	−0.116	0.705	1

[a]See Table 1 for explanation of variable label.

Table 4. Global Fit Indices for Model 1 and Model 2

Index	*Model 1* *Values*	*Model 2* *Values*
X^2_M	17.17	3.90
df_M	15	7
P	0.309	0.791
RMSEA (90% C.I.)	0.027 (0.000−0.076)	0.00 (0.000−0.058)
CFI	0.998	1.00
TLI	0.995	1.01
SRMR	0.015	0.009

(MI = 4.635). Inclusion of a direct path from *ASF* to *clean* and a correlation between *femlit* and *LEB* were not significant ($P > 0.05$) and so were not included in the model. Further evidence of good localized fit was displayed in the standardized residuals, which reflect how well the variances and covariance matrix produced by the model parameters fit the observed variance and covariance matrix. Standardized residuals, which are interpreted as *z*-scores, can be conceived as the number of standard deviations by which the predicted residuals differ from zero-value residuals that would result from a perfectly fitting model (Brown 2006). For model 2, no residuals above the 2.58 significance level were present. For model 1, the sole standardized residual above the 2.58 significance level was a negative residual (−2.78) between *noncomm* and *ASF*. As this residual is negative, it indicates that the model parameters overestimate the observed relationship between *noncomm* and *ASF*. Although significant, this residual is not an outlying value, which may have been indicative of serious model misspecification, as other residuals are close to the 2.58 cut-off point (Brown 2006).

Model Interpretation. Path diagrams representing the predicted relationship between resources, mortality, and fertility are presented in Figures 1 and 2. These figures can be conceptualized as the graphical equivalent of a set of regression equations that relate the dependent and predictor variables (Byrne 2012). Each path tested is indicated by a straight line with a single-headed arrow, which points in the proposed direction of causality. Path coefficients (i.e., the number immediately above or below the single-headed arrow) are standardized and are interpreted as the expected change in SD units of the dependent variable given a 1.00 SD change in the predictor variable, controlling for the direct effects of other variables. The curved double-headed arrows on the left side of the model indicate correlations between pairs of predictor variables. The strength of the correlation between two variables is indicated by the number within the curved double-headed arrow connecting those two variables. The number inside the circles adjacent to each dependent variable indicates the residual variance associated with that dependent variable.

Interpretation of the path coefficients will follow the predicted relationships between resources, mortality, and fertility rates. Predictors of mortality

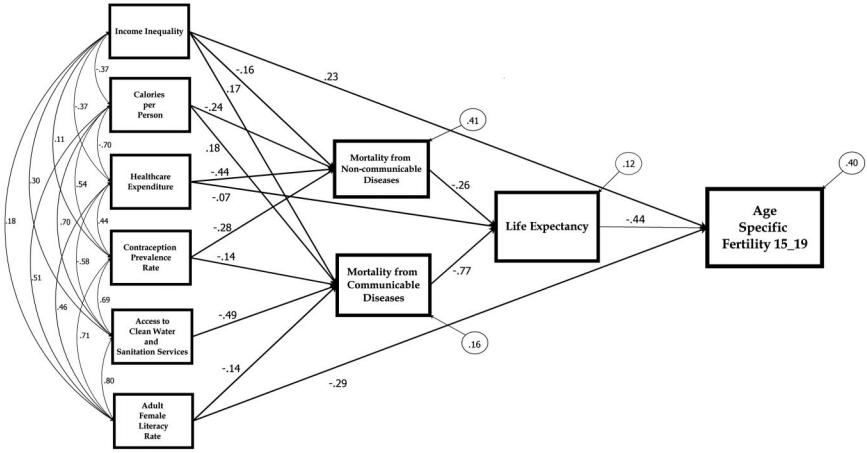

Figure 1. **Model 1.** Numbers associated with single-headed arrows are standardized path coefficients. Numbers associated with curved double-headed arrows are correlations. Numbers within circles are the residuals associated with a dependent variable. All path coefficients are significant at the $P < 0.05$ level.

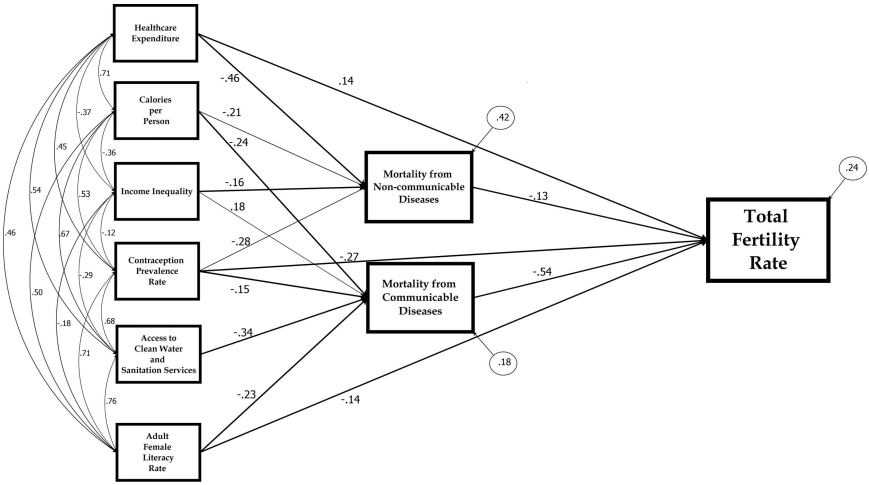

Figure 2. **Model 2.** Numbers associated with single-headed arrows are standardized path coefficients. Numbers associated with curved double-headed arrows are correlations. Numbers within circles are the residuals associated with a dependent variable. All path coefficients are significant at the $P < 0.05$ level with the exception of the path between total fertility rate and adult-female literacy rate, which was approaching significance at $P = 0.077$.

causes will be discussed first followed by predictors of LEB (for model 1), and finally predictors of adolescent and total fertility. Direct effects on a dependent variable are discussed before discussion of the indirect effects (see Data Analysis section for explanation of direct and indirect effects). Standardized path coefficients predicting years lost to communicable diseases, LEB, and adolescent and total fertility rates are translated into original metrics. Adolescent fertility rates are rounded up to the next birth.

Results: Model 1

Mortality Causes. Resource variables accounted for 84% of the variance in years lost to communicable disease and 59% of the variance in years lost to noncommunicable disease (see Table 5). Access to clean water and sanitation services had a strong effect on years lost to communicable diseases with an SD increase predicting a 13.7 decrease in years lost controlling for other resources. Remaining resource variables (i.e., *calories, GINI, CPR, femlit*) exhibited similar effects with SD increases resulting in an approximate 4-year decrease in years lost to communicable disease (see Table 4). The sole exception to this trend was total health-care expenditure, which did not account for a significant portion of variance in years lost to communicable diseases ($P > 0.05$). Total health-care expenditure, however, exhibited the strongest effect on years lost to noncommunicable diseases, with an SD increase, resulting in a -0.45 SD decrease in years lost. Calories per capita and contraception use had similar impacts on years lost to noncommunicable diseases with SD increases predicting an approximate 0.25 SD decrease. GINI exhibited the smallest effect with an SD increase (more inequality) resulting in a 0.16 SD decrease in years lost to noncommunicable diseases.

Life-Expectancy at Birth. Summed across direct and indirect effects, resource variables and mortality causes accounted for 89% of the variance in LEB (see Table 5). Significant direct effects on LEB were produced through total health-care expenditure and years lost to communicable and noncommunicable diseases. SD increases in years lost to communicable and nonommunicable diseases predicted an 8.40-year and a 3.16- year decrease in LEB, respectively. Total health-care expenditure exhibited the smallest direct effect on LEB predicting a 0.75-year decrease. Remaining resource variables had indirect effects on LEB through prior direct effects on years lost to communicable and/or noncommunicable diseases. Access to clean water and sanitation services exhibited the largest indirect effect on LEB with an SD increase, predicting a 4.10-year increase in LEB. SD increases in other resource variables had similar but smaller impacts on LEB with calories per capita and contraception prevalence rate, predicting an approximate 2-year increase and total health-care expenditure an approximate 1-year increase in LEB.

Table 5. Model 1, Effect Decomposition Table[a]

	Dependent			
	COMM	***NONCOMM***	***LEB***	***ASF***
LEB				
Direct Effects				−0.436
Indirect Effects				
Total Effects				**−0.436**
Years lost communicable				
Direct Effects			−0.789	
Total Indirect Effects				0.344
Total Effects			**−0.789**	**0.344**
Years lost noncommunicable				
Direct Effects			**−0.**297	
Indirect Effects				0.130
Total Effects			**−0.297**	**0.130**
Calories per capita				
Direct Effects	−0.184	−0.234		
Indirect Effects			0.215	−0.094
Total Effects	**−0.184**	**−0.234**	**0.215**	**−0.094**
Total health expenditure				
Direct Effects		−0.445	−0.071	
Indirect Effects			0.132	−0.027
Total Effects		**−0.445**	**0.061**	**−0.027**
Clean water and sanitation				
Direct Effects	−0.488			
Indirect Effects			0.385	−0.168
Total Effects	**−0.488**		**0.385**	**−0.168**
GINI (Income Inequality)				
Direct Effects	0.174	−0.157		0.228
Indirect Effects			−0.090	0.039
Total Effects	**0.174**	**−0.157**	**−0.090**	**0.287**
Adult-female literacy rate				
Direct Effects	−0.136			−0.291
Indirect Effects			0.108	−0.047
Total Effects	**−0.136**		**0.108**	**−0.338**
Contraception prevalence				
Direct Effects	−0.144	−0.275		
Indirect Effects			0.196	−0.085
Total Effects	**−0.144**	**−0.275**	**0.196**	**−0.085**

[a]Bolded numbers are the total effects of a variable. See Table 1 for explanation of variable label.

Adolescent Fertility Rates. The final model accounted for 60% of the variance in adolescent fertility rates (see Table 5). Variables with a direct effect on adolescent fertility rates were LEB, the GINI coefficient, and adult-female literacy rates. LEB had the strongest direct effect on adolescent fertility rates with every SD increase associated with a 19-birth decrease per 1,000 adolescent women. The GINI coefficient and adult-female literacy rate also had indirect effects on adolescent fertility rate through prior direct effects on years lost to communicable and/or noncommunicable diseases. Summed across both direct

and indirect effects, SD increases in female literacy rates and GINI predicted 15- and 12-birth decreases, respectively. The impacts of mortality causes (i.e., communicable and noncommunicable diseases) on adolescent fertility were completely mediated through prior direct effects on LEB. An SD increase in years lost to communicable diseases predicted a 15-birth decrease while a smaller indirect effect was produced by years lost to noncommunicable disease with an SD increase predicting a 6-birth decrease. All resource variables had an indirect effect on adolescent fertility rates. The strongest indirect effect on adolescent fertility rate was produced through access to clean water and sanitation with an SD increase predicting a 7-birth decrease. Calories per capita and contraception prevalence rate predicted approximately a 4-birth decrease, respectively. Total health-care expenditure had the smallest effect on adolescent fertility rates, predicting a 3-birth decrease.

Results: Model 2

Mortality Causes. Resource variables accounted for 82% of the variance in years lost to communicable and 58% of the variance in years lost to noncommunicable disease (see Table 6). In general, both the pattern and magnitude of relationships found between resources and mortality causes were similar to model 1. Access to clean water and sanitation services had the strongest direct effect on years lost to communicable diseases with an SD increase predicting a 9.6-decrease in years lost, controlling for the direct effect of other resources. Calories per capita and adult-female literacy rates had a smaller but similar direct effect with SD increases, resulting in an approximate 6-year decrease in years lost to communicable disease. Income inequality and contraception prevalence rates exhibited the smallest direct effects with an SD increase in each, predicting a 5.1-year increase and a 4.3-year decrease in years lost to communicable diseases, respectively. Like model 1, the sole resource variable without a significant direct effect on years lost to communicable diseases was total health-care expenditure. Total health-care expenditure, however, exhibited the strongest effect on years lost to noncommunicable diseases with an SD increase, resulting in an −0.46 SD decrease in years lost. Contraception prevalence rate had the second-largest direct effect with an SD increase, predicting an −0.281 SD decrease in years lost to noncommunicable disease. Calories per capita exhibited the third-largest effect on years lost with an SD increase, predicting an −0.210 SD decrease. Income inequality exhibited the smallest direct effect on years lost to noncommunicable diseases with an SD increase, predicting an −0.156 SD decrease.

Life-expectancy at birth was not included in model 2 as its effect on total fertility rate was completely mediated by years lost to communicable and noncommunicable disease. Additionally, its inclusion did not improve the global or local fit of model 2. Due to the absence of LEB, both years lost to communicable and noncommunicable diseases had direct effects on TFR.

Table 6. Model 2, Effect Decomposition Table[a]

	Dependent		
	COMM	*NONCOMM*	*TFR*
Years lost to communicable disease			
Direct Effects			0.537
Total Indirect Effects			
Total Effects			**0.537**
Years lost to noncommunicable disease			
Direct Effects			0.125
Indirect Effects			
Total Effects			**0.125**
Calories per capita			
Direct Effects	−0.244	−0.210	
Indirect Effects			−0.158
Total Effects	**−0.244**	**−0.210**	**−0.158**
Total healthcare expenditure			
Direct Effects		−0.457	0.136
Indirect Effects			−0.057
Total Effects		**−0.457**	**0.079**
Clean water and sanitation			
Direct Effects	−0.342		
Indirect Effects			−0.184
Total Effects	**−0.342**		**−0.184**
GINI (Income Inequality)			
Direct Effects	−0.180	−0.156	
Indirect Effects			0.077
Total Effects	**−0.180**	**−0.156**	**0.077**
Adult-female literacy rate			
Direct Effects	−0.229		−0.141
Indirect Effects			−0.123
Total Effects	**−0.229**		**−0.264**
Contraception prevalence			
Direct Effects	−0.154	−0.281	−0.269
Indirect Effects			−0.083
Total Effects	**−0.154**	**−0.281**	**−0.352**

[a]Bolded numbers are the total effects of a variable. See Table 1 for explanation of variable label.

Total Fertility Rates. The final model accounted for 76% of the variance in total fertility rates across 191 nations (see Table 6). Again, both the pattern and magnitude of relationships found between predictor variables and fertility were consistent with results from model 1. Variables with a direct effect on total fertility rates were the mortality variables of years lost to communicable and noncommunicable disease and the resource variables of total health-care expenditure, contraception-prevalence rate, and adult-female literacy rate. Across all variables, years lost to communicable disease had the largest impact with an SD increase, predicting a 0.82-year increase in fertility across the reproductive life-span. Contraception-prevalence rate exhibited the second-largest direct effect with an SD increase, predicting a 0.41-year decrease in TFR. The direct effect of adult-female literacy, which was approaching significance ($P = 0.077$), predicted

a 0.22-year decrease in TFR for every SD increase. The effect of total health-care expenditure was of similar magnitude but in the opposing direction with an SD increase, predicting a 0.21-year increase in TFR. Years lost to noncommunicable disease had the smallest direct effect on TFR, with an SD increase, predicting a 0.19-year increase in fertility.

All resource variables, including those with direct effects, had indirect effects on TFR. Indirect effects were produced through a resources prior direct effect on years lost to communicable and/or noncommunicable diseases. Like model 1, the largest indirect effect on total fertility rate was exhibited by access to clean water and sanitation services with an SD increase, predicting a 0.28-year decrease in TFR. Calories per capita had the second-largest indirect effect with an SD increase, predicting a 0.24-year decrease in TFR. An SD increase in adult-female literacy rates predicted a 0.19-year decrease in TFR. The indirect effects of contraception prevalence rate and income inequality were similar in magnitude but in the opposing direction with an SD increase, predicting a 0.13-year decrease and a 0.12-year increase, respectively. Total health-care expenditure had the smallest indirect effect on total fertility rates with an SD increase predicting a 0.09-year decrease in fertility across the female reproductive life-span.

Discussion

By modeling the impact of resources on the risk environment, path analysis allows us to begin partitioning mortality into extrinsic and intrinsic components—a crucial next step in human life-history research. Results from both path models provide strong support for theoretical predictions and largely concur with results of previous studies. Higher population-mortality rates, as reflected by lower life-expectancy at birth (model 1) and greater years lost to communicable and noncommunicable diseases (model 2), are associated with "faster" life-history strategies, as indicated by higher adolescent and total fertility rates. For example, as indicated by model 1, every year decrease in LEB predicts two more births per 1,000 adolescent females Numerous studies have documented this relationship between mortality and fertility; however, the current study models how the availability of resources, through prior impacts on mortality, ultimately affect the timing and frequency of reproduction in humans. In particular, the use of a PA approach enables the test of whether decreases in resources, by mediating an individual's ability to cope with mortality causes, lead to faster reproductive strategies. Both models supported this relationship. In model 1, an SD decrease in all resources, including access to education, health care, clean water and sanitation, calories, contraception and income equality (i.e., GINI), predicted an SD increase in adolescent fertility (SD $\approx$ 44 births/1000 females). Similarly, in model 2, an SD decrease in access to education, clean water and sanitation, calories, and income equality combined to predict 1.58 more births across the female reproductive life-span, slightly more than an SD. While both path models generate a more nuanced representation of the relationship between resources and fertility rates, they also allow for the

quantification of the relative impacts associated with each resource. A resource's relative impact is calculated by division of the standardized total effects (see Tables 5 and 6). In model 1, for example, division of the total effects of female literacy (−0.388) and total health-care expenditure (−0.027) on adolescent fertility reveals that female literacy has a 14- times greater impact on adolescent fertility rates.

In support of the first hypothesis, model 1 clearly demonstrated that mortality causes accounting for the most variance in population mortality rates account for the most variance in adolescent fertility rates. Results of both models also support the predicted role of communicable diseases. Indeed, communicable diseases account for more of the variance in both LEB and both indicators fertility rates. Model 1 indicates that every SD increase in years lost to communicable disease exhibited an almost 3-times greater impact on LEB (8.40 years) compared to noncommunicable diseases (3.16 years). Model 2 also shows that communicable diseases account for more variance in total fertility rates.

Results of both models also supported the second hypothesis: Resources impacting the survival probabilities of communicable diseases—by impacting transmission, prevention, and treatment—have stronger effects on fertility than resources impacting survival probabilities of noncommunicable diseases. Access to clean water and sanitation showed the largest indirect effect of any resource variable on both adolescent. Contraception-prevalence rate had the third-largest indirect effect of all resource variables on adolescent fertility rates. Adult-female literacy rates, which exhibited the third-largest indirect effect on total fertility in model 2, indirectly impact the disease transmission environment as literate females may be more educated in disease prevention (e.g., sex education).

Our results, in general, concur with predictions from LHT and previous studies. However, a few predicted relationships were not found. In both models total health-care expenditure did not have a significant impact on years lost to communicable disease. This finding may indicate that total health-care expenditure is not a strong indicator of access to health services that specifically target communicable diseases, or a majority of health-care funds are allocated to the treatment of noncommunicable diseases. In the majority of developed nations, communicable diseases with the potentially greatest impact on life-expectancy (i.e., diseases that affect childhood mortality) have either been eradicated through large-scale immunization programs (e.g., typhoid, cholera, and tuberculosis) or by tactics and infrastructure improvements that decrease transmission rates (e.g., mosquito-prevention programs). As a result of these measures, the mortality rates in developed countries are less impacted by communicable diseases (World Health Organization 2009). Because the extension of adult-life expectancy increases age-related noncommunicable diseases (e.g., cancer and heart disease) that are expensive to treat (Narayan, Ali, and Koplan 2010), the strong relationship between total health-care expenditure and noncommunicable diseases in developed countries may disguise the effect of total health-care expenditure on communicable diseases in developing countries. Alternatively,

health-care funding in developing countries may not produce a significant decrease in mortality from communicable diseases for many years. This reasoning may partly explain the unexpected result in model 2 where total health-care expenditure predicted an increase in total fertility rates. Future studies should incorporate indicators of health-care funding with direct relationships to communicable diseases (e.g., access to immunization programs and STI prevention education).

Some resources, including adult-female literacy rate, contraception-prevalence rate and income inequality, affect fertility rates largely outside the context of mortality. Female literacy and income inequality produced the smallest indirect effects on LEB in model 1, but were the only resource variables with direct effects on adolescent fertility. As such the effects of education and income inequality on LEB may need to be more closely evaluated. Another possibility may be that a threshold level of education must be reached before effects on LEB are statistically noticeable (Bulled and Sosis 2010). More difficult to explain is the weak effect of income inequality on LEB. The likely consequences of income inequality on access to health care and overall standard of living suggest that variation in population LEB should be intimately tied with the GINI coefficient. However, inequality may interact with other variables in complex ways not detectable in a global comparison.

In summary, on a global scale, resources influence life-history largely through their impact on communicable diseases. However, in populations where disease burdens have been substantially reduced, then other indicators of extrinsic and intrinsic risk come into play. A promising line of research indicates that in healthy populations psychosocial stress apparently tunes life-history development in ways similar to mortality in less developed populations (Chisholm and Coall 2008).

Conclusion

Data from 191 countries were used to test two hypotheses operating as separate links in the causal chain from resources to fertility rates. Results of two path models confirm that resources with the greatest impact on the survival associated with communicable diseases have the greatest impact on the timing and frequency of reproduction. A PA approach generates a more nuanced representation of the direct and indirect relationships operating between resources and reproductive behavior. This approach suggests that some environmental factors, such as communicable versus noncommunicable disease, appear to have effects more like extrinsic risks versus intrinsic risks. While this study does not entirely resolve important issues in isolating mortality sources, it does improve our understanding of how local conditions' influence life-history strategies. Hence, this approach may prove useful in new theory development and in population planning.

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Appendix

Appendix A. Relationships between Indicators of Fertility

Table 7. Correlations among Fertility Rates (asf) at Every Age Cohort and Total Fertility Rate (TFR)[a]

	asf15_19	*asf20_24*	*asf25_29*	*asf30_34*	*asf35_39*	*asf40_44*	*asf45_49*	*TFR*
asf15_19	1							
asf20_24	0.85	1						
asf25_29	0.68	0.89	1					
asf30_34	0.62	0.78	0.96	1				
asf35_39	0.63	0.80	0.93	0.98	1			
asf40_44	0.65	0.80	0.89	0.93	0.97	1		
asf45_49	0.54	0.70	0.79	0.81	0.86	0.90	1	
TFR	0.78	0.92	0.96	0.95	0.95	0.94	0.84	1

[a]Correlations are calculated from raw values.

Appendix B. Relationships between Variables and Population Size of Country

Table 8. Correlations between Country Population Size and Variables. Across All Variables Only Contraception Prevalence Rate Has a Significant Correlation with Population Size ($r = 0.172$ $P < 0.05$)[a]

	TFR	*Comm*	*Noncomm*	*Calorie*	*Health*	*Clean*	*GINI*	*CPR*	*Femlit*
population	−0.052	−0.005	−0.024	0.057	−0.027	−0.028	0.091	0.172*	−0.049

[a]Correlations are calculated from raw values. See Table 1 for explanation of variable label.

Appendix C. Proportion of Data Present

Table 9. Covariance Coverage Matrix for Model 1 Indicates the Proportion of Raw Data Present for Each Variable and Pairs of Variables Prior to Estimation[a]

	ASF	*LEB*	*Comm*	*Noncomm*	*Calorie*	*Health*	*Clean*	*GINI*	*CPR*	*Femlit*
ASF	0.921									
LEB	0.916	0.921								
Comm	0.916	0.916	0.995							
Noncomm	0.916	0.916	0.995	0.995						
Calorie	0.859	0.859	0.885	0.885	0.890					
Health	0.901	0.901	0.974	0.974	0.874	0.979				
Clean	0.874	0.874	0.932	0.932	0.853	0.921	0.937			
GINI	0.874	0.874	0.901	0.901	0.848	0.890	0.864	0.906		
Femlit	0.895	0.890	0.916	0.916	0.848	0.901	0.869	0.869	0.921	
CPR	0.796	0.801	0.848	0.848	0.775	0.832	0.806	0.785	0.785	0.853

[a]See Table 1 for Description of Variable Label.

Table 10. Covariance Coverage Matrix for Model 2 Indicates the Proportion of Raw Data Present for Each Variable and Pairs of Variables Prior to Estimation[a]

	TFR	*Comm*	*Noncomm*	*Calorie*	*Health*	*Clean*	*GINI*	*CPR*	*Femlit*
TFR	1.000								
Comm	0.995	0.995							
Noncomm	0.995	0.995	0.995						
Calorie	0.885	0.880	0.880	0.885					
Health	0.979	0.974	0.974	0.869	0.979				
Cleansani	0.937	0.932	0.932	0.848	0.921	0.937			
GINI	0.906	0.901	0.901	0.843	0.890	0.864	0.906		
CPR	0.853	0.848	0.848	0.775	0.832	0.806	0.785	0.853	
Femlit	0.927	0.916	0.921	0.848	0.901	0.874	0.874	0.791	0.927

[a]See Table 1 for description of variable label.

Appendix D. Raw Data

Table 11. Raw Data[a]

Country	*TFR*	*ASF*	*LEB*	*Comm*	*Noncomm*	*Cal*	*CS*	*Femlit*	*GINI*	*Health*	*CPR*
Afghanistan	7.1	125	43	77	1,309	–	25.5	12.6	60	91	10
Albania	2.1	14	76	12	752	2,875	93	98.3	33	239	60
Algeria	2.4	8	72	43	565	2,928	90.5	60.1	35.3	188	61
Andorra	1.3	–	–	7	373	–	100	99.9	–	1,905	–
Angola	6.5	127	46	81	1,071	1,902	42	54.2	58.6	56	6
Antigua and Barbuda	2.1	–	–	17	674	2,378	93	–	57.3	599	53
Argentina	2.3	58	75	18	515	3,180	92.5	97.2	50	814	–
Armenia	1.4	36	73	13	1,064	2,006	91	99.2	33.8	130	53
Australia	1.8	15	80	6	355	3,110	100	99	35.2	2,271	–
Austria	1.4	13	79	4	409	3,794	100	99.9	29.1	2,858	51
Azerbaijan	1.8	34	67	37	856	2,387	78	98.2	36.5	104	51
Bahamas	2	54	72	36	509	2,736	98.5	96.5	57	1,361	–
Bahrain	2.3	17	75	12	678	–	–	83.6	–	820	62
Bangladesh	2.9	76	65	61	730	2,158	55.5	41.4	33.4	27	56
Barbados	1.5	43	76	22	531	2,946	100	99.7	39	916	55
Belarus	1.2	22	69	5	854	2,895	96	99.4	29.7	328	73
Belgium	1.6	8	79	5	437	3,695	100	99	33	2,519	78
Belize	3	81	76	33	677	2,867	69	77.1	51	229	34
Benin	5.5	113	60	78	835	2,537	44	23.3	36.5	50	17
Bhutan	2.2	43	65	57	708	–	66.5	34	46.8	132	35
Bolivia	3.5	79	65	54	765	2,228	60.5	80.7	58.2	282	61
Bosnia and Herzegovina	1.2	17	75	6	670	2,723	96.5	94.4	26.2	282	36
Botswana	2.9	54	49	84	594	2,256	70	81.8	61	374	48
Brazil	2.3	78	72	30	625	3,002	81.5	88.8	55	506	81
Brunei Darussalam	2.3	26	77	16	473	2,758	–	90.2	–	1,036	–
Bulgaria	1.3	43	73	5	733	2,544	99	97.7	29.2	377	86
Burkina Faso	6	132	52	82	924	2,439	32.5	15.2	39.6	41	17
Burundi	6.8	21	49	80	919	1,604	56.5	52.2	42.4	12	9
Cambodia	3.2	41	58	67	832	2,011	27	64.1	40.7	51	40
Cameroon	4.4	129	50	78	840	2,254	55	59.8	44.6	75	29
Canada	1.5	13	80	6	374	3,178	100	99	32.6	2,514	75
Cape Verde	3.4	96	70	53	591	3,286	60.5	69.2	50.5	97	61
Central African Republic	4.6	110	44	78	868	1,968	42.5	33.5	43.6	25	19
Chad	6.2	169	51	82	910	2,083	20.5	12.8	39.8	49	3
Chile	1.9	60	78	10	458	2,867	92	95.6	54.9	572	58
China	1.7	10	73	20	627	2,979	69.5	86.5	46.9	109	85
Colombia	2.2	79	72	22	483	2,576	82.5	90.7	58.5	370	78
Comoros	4.4	47	64	66	713	1,764	58.5	49.3	64.3	21	26
Congo	4.5	207	53	79	716	2,236	45	78.4	47.3	56	44
Cook Islands	2.6	–	–	29	570	–	97.5	–	–	436	44
Costa Rica	2.1	69	79	14	439	2,749	96.5	95.1	49.8	467	96
Côte d'Ivoire	4.5	131	57	74	559	2,588	48.5	38.6	44.6	84	13
Croatia	1.3	14	75	5	578	2,597	99	97.1	29	839	–
Cuba	1.5	46	78	9	437	2,614	94.5	99.8	40	353	77
Cyprus	1.6	7	79	9	412	3,283	100	96.3	29	1,973	.
Czech Republic	1.2	11	76	4	559	3,028	99.5	99	25.8	980	69
Dem. People's Rep. of Korea	1.9	–	67	40	642	2,165	79.5	–	31	1	81
Dem. Rep. of the Congo	6.7	–	46	81	921	1,557	35	74.9	44.4	8	21
Denmark	1.8	6	78	4	495	3,443	100	99.9	24.7	2379	–
Djibouti	4	24	54	72	862	2,182	74	58.4	40	90	23
Dominica	2.1	–	–	20	580	2,991	90	94	49	387	50
Dominican Republic	2.8	109	72	40	794	2,319	83	87.2	50	333	73
Ecuador	2.6	83	75	34	484	2,726	84	89.7	54.4	202	73
Egypt	2.9	41	70	31	891	3,376	79	59.4	34.4	208	60
El Salvador	2.7	85	71	37	518	2,470	80.5	77.7	52.4	351	73
Equatorial Guinea	5.4	124	50	78	938	–	47	80.5	39	160	–
Eritrea	5.1	70	57	73	686	1,669	29	47.6	–	33	8

(Table Continues)

Table 11. (*continued*)

Country	*TFR*	*ASF*	*LEB*	*Comm*	*Noncomm*	*Cal*	*CS*	*Femlit*	*GINI*	*Health*	*CPR*
Estonia	1.5	22	73	5	664	2,946	97.5	99.8	36	521	70
Ethiopia	5.3	105	54	82	817	1,887	18	35.1	29.8	19	15
Fiji	2.8	34	68	24	767	2,778	58.5	91.9	50	160	35
Finland	1.8	12	79	4	405	3,169	100	99.9	26.9	1,794	–
France	1.9	7	80	6	387	3,597	100	99	32.7	2,542	75
Gabon	3.1	93	60	68	716	2,585	60.5	53.3	41.5	552	33
Gambia	4.8	92	55	72	830	2,273	67.5	32.8	50.2	39	18
Georgia	1.4	45	71	25	554	2,236	90	99.9	40.8	153	47
Germany	1.4	8	79	5	429	3,506	100	99	28.3	2,670	75
Ghana	3.9	66	57	73	699	2,613	40.5	49.8	40.8	65	24
Greece	1.3	9	79	4	436	3,738	98.5	94.2	34.3	1,449	–
Grenada	2.3	44	68	26	827	2,758	95.5	–	45	388	54
Guatemala	4.2	109	70	51	515	2,148	85.5	63.3	53.7	217	43
Guinea	5.5	155	56	77	844	2,320	38.5	18.1	43.3	47	9
Guinea-Bissau	7.1	129	47	83	925	2,486	44	27.4	35.5	34	10
Guyana	2.3	64	66	41	835	2,639	85.5	98.5	43.2	116	34
Haiti	3.6	48	60	67	740	2,046	40	51.2	59.5	61	32
Honduras	3.3	95	70	47	761	2,394	69	80.2	53.8	138	65
Hungary	1.3	21	73	3	693	3,552	99.5	99.3	26.9	852	77
Iceland	2	16	81	4	375	3,214	100	99	25	2738	–
India	2.8	70	64	56	713	2,489	52.5	47.8	36.8	63	56
Indonesia	2.2	41	70	31	690	2,913	64.5	86.8	34.3	37	61
Iran (Islamic Republic of)	2	20	70	28	687	2,935	88.5	70.4	43	387	79
Iraq	4.3	82	68	42	1,018	–	76	64.2	42	84	50
Ireland	2	17	79	7	459	3,701	100	99	34.3	1,950	–
Israel	2.8	15	80	9	368	3,510	100	95.9	39.2	1,845	–
Italy	1.4	5	80	5	372	3,663	100	98	36	2,061	60
Jamaica	2.5	79	72	35	605	2,686	88	91.6	45.5	313	69
Japan	1.3	5	82	8	284	2,753	100	99	38.1	1,967	56
Jordan	3.1	25	72	29	711	2,732	93.5	84.7	37.7	302	57
Kazakhstan	2.3	30	66	25	1,145	2,386	96.5	99.3	33.9	198	51
Kenya	5	104	53	82	729	2,037	46	79.7	42.5	51	39
Kiribati	4.1	–	61	42	730	2,910	46	–	–	154	22
Kuwait	2.2	13	78	13	454	3,151	–	91	30	903	50
Kyrgyzstan	2.5	32	68	35	1,012	2,877	87.5	98.1	32.9	62	48
Lao People's Dem. Republic	3.2	40	64	62	828	2,303	34	60.9	34.6	41	38
Latvia	1.3	15	71	5	710	2,720	88.5	99.8	35.7	456	48
Lebanon	2.2	17	72	20	715	3,151	99	82.2	45	801	58
Lesotho	3.4	77	43	86	581	2,304	55.5	94.5	52.6	65	37
Liberia	6.8	142	57	84	931	2,176	47.5	41.6	–	14	11
Libyan Arab Jamahiriya	2.8	3	74	29	654	3,324	84	72	35.8	385	45
Lithuania	1.3	23	71	5	635	3,293	–	99.6	36	543	47
Luxembourg	1.7	13	79	7	419	–	100	99.9	26	3,137	–
Madagascar	4.8	136	59	74	799	2,138	28	62.5	47.5	21	27
Malawi	5.6	140	47	87	796	2,166	59	49.8	37.9	38	41
Malaysia	2.6	13	74	28	623	2,917	96	85.4	49.2	289	55
Maldives	2.6	14	67	35	953	2,552	72.5	96.4	39	170	39
Mali	6.5	163	53	83	967	2,358	46.5	39.6	40.1	52	8
Malta	1.4	12	80	6	433	3,543	–	93.6	28	2,864	–
Marshall Islands	3.8	–	–	34	961	–	84.5	93.7	–	580	45
Mauritania	4.4	91	63	73	812	2,762	36	43.4	39	40	9
Mauritius	1.9	39	72	10	731	2,989	97	80.5	48.1	302	76
Mexico	2.2	66	74	25	501	3,154	84.5	89.6	46.1	507	71
Micronesia (Fed. States of)	3.8	27	68	32	682	–	59	–	–	216	–
Monaco	1.8	–	–	7	321	–	–	99	33	4,377	–
Mongolia	1.9	15	66	32	923	2,084	58	97.5	32.8	108	66
Morocco	1.8	19	71	39	655	2,966	72.5	39.6	39.5	109	63
Mozambique	2.4	155	43	81	777	1,939	34	32.7	47.3	21	16
Myanmar	5.2	19	61	56	775	2,806	65	–	40	11	34
Namibia	2.1	76	52	82	513	2,743	56.5	83.5	74.3	243	55
Nauru	3.2	–	–	24	1,093	–	–	–	–	940	36

(Table Continues)

Table 11. (*continued*)

Country	*TFR*	*ASF*	*LEB*	*Comm*	*Noncomm*	*Cal*	*CS*	*Femlit*	*GINI*	*Health*	*CPR*
Nepal	3	104	63	60	769	2,446	51.5	34.9	47.3	40	48
Netherlands	3.3	4	79	6	425	3,336	100	99	30.9	2,337	79
New Zealand	1.7	23	80	5	398	3,211	100	99	36.2	1,686	75
Nicaragua	2	114	72	39	705	2,223	61.5	67.8	43.1	133	72
Niger	2.8	169	56	86	1030	2,121	23	15.1	43.9	16	11
Nigeria	7.2	127	47	81	909	2,743	38.5	60.6	43.7	59	15
Niue	5.4	–	–	33	595	–	100	–	–	496	23
Norway	1.8	9	80	4	391	3,338	100	99.9	25.8	3,039	–
Oman	3	11	75	16	664	–	84.5	73.5	32	461	32
Pakistan	3.5	46	65	64	717	2,456	68	36	30.6	40	30
Palau	2.5	–	69	29	735	–	77.5	–	25	1,046	17
Panama	2.6	84	75	35	417	2,215	80.5	91.2	54.9	560	–
Papua New Guinea	3.8	58	57	65	772	2,177	41.5	50.9	50.9	64	32
Paraguay	3.1	74	71	33	602	2,544	68	93	58.4	309	79
Peru	2.5	56	73	41	534	2,599	73	89.4	49.6	232	71
Philippines	3.3	46	71	44	620	2,375	81	92.7	44	80	51
Poland	1.2	14	75	4	583	3,401	100	99.7	34.9	583	49
Portugal	1.5	17	78	9	456	3,757	98	91.3	38.5	1,509	–
Qatar	2.7	17	75	17	512	–	100	88.6	39	1,259	43
Republic of Korea	1.2	5	78	6	470	3,093	–	99	31.6	747	41
Republic of Moldova	1.4	35	68	10	963	2,628	85	98.6	35.6	86	68
Romania	1.3	32	72	9	706	3,329	79	96.3	31.5	320	70
Russian Federation	1.3	26	65	8	904	2,918	91.5	99.2	39.9	410	–
Rwanda	5.9	38	48	83	878	2,058	45	64.7	46.8	24	36
Saint Kitts and Nevis	2.3	–	–	27	691	3,095	97.5	–	42.6	541	54
Saint Lucia	2.2	62	74	17	522	2,958	93.5	90.6	42.6	429	47
Saint Vincent and Grenadines	2.2	60	71	31	674	2,642	–	99	56	282	–
Samoa	4	30	71	32	766	–	94.5	99.4	–	155	43
San Marino	1.3	–	–	5	357	–	–	–	–	2,870	–
Sao Tome and Principe	3.9	69	65	71	788	2,484	52	77.9	–	–	30
Saudi Arabia	3.4	27	73	24	678	2,837	–	70.8	39.2	692	32
Senegal	4.7	105	55	74	852	2,270	49.5	29.2	41.3	54	12
Serbia and Montenegro	1.8	23	73	–	–	2,660	96	94.1	30	411	41
Seychelles	1.7	.	72	17	650	2,437	–	92.3	–	742	–
Sierra Leone	6.5	128	46	83	1,033	1,904	34.5	24.4	42.5	17	8
Singapore	1.3	5	80	12	345	–	–	88.6	42.5	1,151	62
Slovakia	1.2	21	74	5	628	2,789	100	99.7	25	603	74
Slovenia	1.3	6	78	4	480	3,149	–	99.8	28.4	1,447	74
Solomon Islands	3.9	44	63	50	694	2,221	50.5	–	–	80	27
Somalia	6.1	70	47	72	1,148	–	22	25.8	30	–	15
South Africa	2.7	61	51	69	867	2,908	73	85.7	57.8	519	60
Spain	1.4	12	81	7	379	3,387	100	97.2	34.7	1,536	81
Sri Lanka	1.9	30	72	8	681	2,345	79	89.1	40.2	99	68
Sudan	4.3	59	57	57	986	2,272	51.5	50.5	51	37	8
Suriname	2.4	41	69	31	728	2,625	87	87.2	52.9	369	46
Swaziland	3.5	88	46	83	707	2,541	54.5	80.8	50.4	207	51
Sweden	1.8	8	81	5	372	3,100	100	99	25	2,283	–
Switzerland	1.4	6	81	5	360	3,435	100	99	33.7	3,265	82
Syrian Arab Republic	3.1	64	74	25	679	3,052	86.5	73.6	42	159	58
Tajikistan	3.4	29	66	72	884	1,716	72.5	99.2	32.6	41	37
TFYR Macedonia	1.4	23	74	6	737	2,695	89.5	94.1	39	470	–
Thailand	1.8	39	69	42	516	2,459	95	90.5	42.5	172	77
Togo	4.9	67	62	78	818	2,281	33.5	46.9	34.4	32	17
Tonga	3.8	23	72	31	658	–	98	99	47	163	23
Trinidad and Tobago	1.6	35	69	26	751	2,713	91.5	98	40.3	–	43
Tunisia	1.9	7	74	41	537	3,310	85.5	65.3	39.8	271	60
Turkey	2.1	40	71	26	701	3,374	90	79.6	43.6	432	73
Turkmenistan	2.5	20	63	48	1100	2,715	–	98.3	40.8	–	48
Tuvalu	3	–	–	30	979	–	89.5	–	–	324	31

(Table Continues)

Table 11. (*continued*)

Country	*TFR*	*ASF*	*LEB*	*Comm*	*Noncomm*	*Cal*	*CS*	*Femlit*	*GINI*	*Health*	*CPR*
Uganda	6.5	154	51	80	786	2,382	44	57.7	45.7	45	24
Ukraine	1.2	29	68	9	881	2,898	96.5	99.2	28	198	67
United Arab Emirates	2.3	17	79	18	410	3,333	98.5	81.7	31	1,263	28
United Kingdom	1.8	25	79	7	441	3,312	100	99	36	1,846	84
United Republic of Tanzania	5.2	131	54	79	851	1,958	43.5	62.2	34.6	30	26
United States of America	2.1	37	78	9	450	3,814	99.5	99	46.2	4,570	76
Uruguay	2.1	62	76	12	521	2,838	100	98.4	44.9	818	–
Uzbekistan	2.5	13	–	48	880	2,286	91.5	99	36.8	83	65
Vanuatu	3.8	49	69	39	749	2,583	54.5	74	–	127	38
Venezuela	2.6	90	73	21	441	2,360	–	92.7	48.2	510	77
Viet Nam	2.2	17	74	39	611	2,498	64	86.9	37.7	75	76
Yemen	5.5	70	62	60	941	2,041	54.5	30	33.4	84	28
Zambia	5.2	146	44	85	833	1,901	51.5	74.8	50.8	52	41
Zimbabwe	3.2	67	43	85	816	2,104	62.5	95.7	54	1	60

[a]See Table 1 for description of variable label.