Parents' Age at Birth of Their Offspring and Child Survival

Guillaume Wunsch and Catherine Gourbin

Institute of Demography, University of Louvain

ABSTRACT: This study presents some new results on parental age as a risk factor for child survival. The study is based on individual registration forms for live births and infant deaths collected in Hungary from 1984 to 1988. Logistic regression models have been fitted for early neonatal and neonatal mortality on the one hand, and post-neonatal mortality on the other hand. Children of older males and females have significantly higher early neonatal and neonatal mortality rates compared to those of younger males and females. The impact of age of both parents remains, however, slighter than that of other biological characteristics such as previous number of fetal deaths, induced abortions, or live births. The authors discuss possible biological explanations.

Initially, most programs and scientific studies in the field of reproductive health have rightly focused on women, as either teenage or adult mothers. An important development has been the adoption of a gender perspective in the study of reproductive health. Following this approach, it is now recognized that men, too, have to be taken into account in research-action programs on reproductive health. Males are particularly involved, among other things, in family decisions relating to the tempo and quantum of fertility, and in the knowledge and use of contraception. Moreover, the impact of males on the intermediate fertility variables, such as frequency of intercourse, has to be taken into account. Similarly, studies pertaining to infant and child mortality differentials should consider both maternal and paternal characteristics.

Infant and child mortality has usually been studied in demography according to the characteristics of the mother. The literature shows an impact of the social and biological characteristics of the mother on fetal mortality and child survival (Rish et al., 1988; Gourbin, 2002; Masuy-Stroobant, 1997), such as age of the mother, previous number of fetal deaths, and education. In particular it is the mother who is in charge of prenatal care and who in most cases will be in charge of the rearing of the child. Several studies have also considered the socioeconomic characteristics of the father. It is well known, for example, that the father's social class is significantly associated with infant mortality in England and Wales (Macfarlane and Mugford, 1984; Haines, 1995). The education and occupation of the father have also been addressed by various studies in low-fertility countries, such as those of Masuy-Stroobant (1988) and Arntzen et al. (1993). Moreover, in high-fertility countries, various studies have shown that the level of education of the father, for example, is slightly less or as important as that of the mother in relation to child survival (Trussell and Hammerslough, 1983; Akoto and Tabutin, 1989; Majumder et al., 1997). Very few studies have, however, dealt with the impact of paternal age at conception on infant and child survival and health. This is all the more surprising in that several countries, such as Belgium, Hungary, the Czech Republic, and the U.S. indeed have adequate data sets for this purpose but have rarely used them.

The purpose of this paper is thus to examine the respective impact of both age of mother and age of father on infant mortality, controlling for one when examining the other, and taking into account various confounding factors such as parity, number of previous fetal deaths, and number of induced abortions. Though associated with maternal age, these variables have an effect *per se* on subsequent feto-infant mortality (Golding, 1991; Miller, 1989; Leridon, 1987; Bross and Shapiro, 1982; W.H.O., 1979; W.H.O., 1970). They may, however, also partly reflect differences in socioeconomic status. We have therefore controlled for the following variables: educational level of mother and father, activity, and type of activity of the mother. Education is related to the adoption of healthy habits during pregnancy and child care (Caldwell, 1979). Thanks to legislative measures to protect mothers during pregnancy, working women benefit from certain rights and are better informed about prenatal care facilities (Saurel-Cubizolles and Kaminski, 1986; Dermont et al., 1990). However, in order to take account of the fact that manual work may lead to higher risks of infant morbidity and mortality, we have included type of activity, distinguishing between manual and non-manual female workers. Activity of the father has not been taken into account, as nearly all fathers were active. In addition, as information on type of activity of father included too many missing cases, we have excluded this variable from our analysis.

BACKGROUND

From the literature, one can point out the following studies dealing with both parental ages in their relationship with fetal and infant mortality. Woolf (1965, cited in Golding (1991)), using data on Arkansas from 1958 to 1961, detected both a maternal and a paternal age effect on stillbirth. Selvin and Garfinkel (1976) have shown, moreover, on the basis of data from the State of New York (1959 - 1967), that age of father and age of mother have a rather similar impact on the occurrence of intra-uterine deaths recorded during the 20th gestation week or after. On the other hand, Resseguie (1976) found no paternal effect on stillbirth using data on Wisconsin from 1968 to 1971, controlling for mother's age and education. A Greek study (Tzoumaka-Bakoula et al., 1989) found an increasing perinatal mortality with advancing paternal age, but this association disappeared once age of mother was included. Another study, by Spira et al. (1993), using data from an *in vitro* fertilization study, found no relationship between the percentage of spontaneous abortions and age of father. The same study shows, on the contrary, spontaneous abortions increasing significantly after age 35 for mothers. As J. Golding (1991) has rightly stressed, the problem with most of these results is the small number of cases involved in the studies. Moreover, the results can be inconclusive due to the extensive confounding between age of mother and age of father. It is therefore necessary to deal with very large samples taking into account both parental ages and, if possible, other confounding variables.

The present paper continues on previous work by Gourbin and Wunsch (1999). In order to go beyond the limitations of previous research, this work was based on very large data sets concerning births and infant deaths derived from the civil register. The vital statistics data were those of Belgium and Hungary. Controlling only for the age of the mother, a statistically significant relationship between age of father and infant mortality was found. In Hungary this relationship remains valid for the infant mortality period as a whole (first year of life), and in Belgium it has also been found for stillbirths (no data available for Hungary), though not for the post-neonatal period. For Hungary, it has also been possible to include in the analysis a series of other co-variables relating to the mother in addition to age, such as previous fetal deaths, previous induced abortions, previous live births, education level, activity, and type of activity. These last three variables were also available for the father. It has been shown that Hungarian children of older fathers have significantly higher mortality rates than those of younger fathers during the first year of life considered as a whole. The impact of age remains, however, slighter than that of other characteristics, such as no previous fetal deaths or no previous induced abortions. It is also interesting to note that the impact of age of mother is not very different from that of age of father once confounding factors are controlled for.

MATERIAL AND METHODS

The present paper examines whether there is a significant association between age at infant death, on the one hand, and age of both parents, on the other hand, this time breaking down the infant mortality period into early neonatal, neonatal, and post-neonatal mortality. Furthermore, as Gavrilov and Gavrilova (1997; 2001b) have shown that at older ages, paternal age at reproduction has a life-shortening effect on daughters rather than sons, we have tested the assumption that there is a differential effect of age of parents at reproduction on the survival of their offspring according to the sex of the child. Individual linked registration forms for live births and infant deaths collected in Hungary from 1984 to 1988 were used here. Only married women have been retained as age of the father is unknown for most unmarried women. It should be stressed that a full maternity history for each woman is not available. For example, we cannot detect if an early child death has led to a replacement birth, which would then be of higher parity. In order to deal with sufficiently large numbers, ages of parents have been grouped in ten-year periods (15–24; 25–34; 35–44), though an analysis with five-year age-groups confirmed our results with wider confidence intervals.

A logistic regression model (see, e.g., Menard, 1995) using the logistic procedure of the SAS Program (SAS Institute, 1989) has been applied to the individual data in order to take all the risk factors into consideration together. The logistic model is equivalent to a discrete-time hazard model. The hazard rate and the effects of covariates are modeled by logistic regression as follows:

$$\text{logit}(\pi_{ti}) = \log(\pi_{ti} / (1 - \pi_{ti})) = \alpha(t) + \Sigma\, \beta_p x_{pti}$$

where π_{ti} is the probability that the i^{th} child dies at age t, x_{pti} is the pth covariate for the ith child at age t, $\alpha(t)$ is the baseline age effect, and the β_p are unknown regression coefficients to be estimated. The model is fitted by maximum likelihood. The dependent variable in this case is either survival or death of the child during the early neonatal, neonatal, or post-neonatal period, while the explanatory variables relate to the characteristics of his or her mother and father. Furthermore, in order to avoid right-censoring of the data, we have excluded the children born after the first trimester of 1988.[1] This has led to a loss of slightly fewer than four hundred infant deaths from the data. Finally, we are left in the present case with about 490,000 live births and about 8,300 infant deaths. The definitions and codings of the covariates of infant mortality are presented in Table 1.

RESULTS

Main effects models have first been fitted, the dependent variable being the probability of dying during the early neonatal period (first 7 days of life), the neonatal period (first 28 days of life), and the post-neonatal period (the rest of the year). The parameters of the model yield the relative impact on the logit of the probability of dying of being in one category of an explanatory variable compared to the reference category. All models fit well according to the G and Score statistics. The following comments are based on a p value ≤ 0.05. As early neonatal and neonatal mortality data lead, for all practical purposes, to the same conclusions, only results concerning neonatal mortality are presented here.

One sees that children from older men and women have significantly higher neonatal mortality rates compared to those from the age group 25–34 (Table 2). It is interesting to note that the impact of age of mother is not very different from that of the age of father once confounding factors are controlled for. The impact of age, however, remains slighter than that of other characteristics, such as no previous fetal deaths or no previous induced abortions. Among the other explanatory variables, one can see that having had one previous live birth has a positive impact on child survival. Low education of the mother has, on the contrary, a detrimental impact on child survival. The same can be said when the mother is a manual worker.

For the post-neonatal period (Table 3), no significant impact of age of mother and father is observed this time. On the other hand, having had no previous live birth, induced abortion, or fetal death increases the chance of survival; the same is true for active mothers as compared to housewives. Low levels of education for mother or father have the opposite effect.

In order to see if age of parents has a differential impact according to sex of child, we have introduced the latter variable and two interaction terms between age of parents and sex of child into the equation. Though the introduction of interaction terms leads to problems of multicolinearity, a strong interaction has been detected between age of father 15–24 and female child. To examine this point further, we have applied the logistic model for neonatal mortality separately to male infants and to female infants. Tables 4 and 5 show the main effects of the explanatory variables for boys on the one hand and for girls on the other hand. One notices that for boys (Table 4), older fathers lead to higher neonatal mortality risks compared to all the younger ages. For girls (Table 5), this relative difference is not observed for fathers in the youngest

[1] This leads to losing the data for the last half of the fourth trimester only, of the first year of life, for those born during the first trimester of 1988. As the number of deaths concerned is very small, this should have no impact on our results.

TABLE 1

DEFINITIONS AND CODING OF COVARIATES OF INFANT MORTALITY IN HUNGARY

Variable Name	Coding Categories	Reference Category
Age of mother	1 = 15–24; 2 = 25–34; 3 = 35–44	35–44
Age of father	1 = 15–24; 2 = 25–34; 3 = 35–44	35–44
Previous live birth(s)	0 = 0 births; 1 = 1 birth; 2 = 2+ births	2+ births
Previous fetal death(s)	0 = 0 deaths; 1 = 1 death; 2 = 2+ deaths	2+ deaths
Previous induced abortion(s)	0 = 0 abortions; 1 = 1 abortion, 2 = 2+ abortions	2+ abortions
Activity of the mother	1 = active; 2 = housewife	housewife
Type of activity of the mother	1 = manual; 2 = non-manual	non-manual
Education of the mother	1 = without + technical; 2 = secondary + university	secondary + university
Education of the father	1 = without + technical; 2 = secondary + university	secondary + university

TABLE 2

ODDS RATIOS FROM DISCRETE-TIME LOGISTIC REGRESSION MODEL OF NEONATAL MORTALITY AMONG CHILDREN OF MARRIED WOMEN IN HUNGARY 1984–1988[1]

Characteristics	Modalities and Reference Group	OR	95% Confidence Limits	p
Age of mother (in years)	15–24 / 35–44	0.76	0.68, 0.86	0.0001
	25–34 / 35–44	0.77	0.70, 0.86	0.0001
Age of father (in years)	15–24 / 35–44	0.88	0.79, 0.97	0.0122
	25–34 / 35–44	0.83	0.76, 0.89	0.0001
Previous live birth(s) (LB)	No pr. LB / 2+ pr. LB	0.78	0.72, 0.85	0.0001
	One pr. LB / 2+ pr. LB	0.72	0.67, 0.78	0.0001
Previous fetal death(s) (FD)	No pr. FD / 2+ pr. FD	0.36	0.33, 0.39	0.0001
	One pr. FD / 2+ pr. FD	0.56	0.50, 0.62	0.0001
Previous induced abortion(s) (IA)	No pr. IA / 2+ pr. IA	0.48	0.44, 0.53	0.0001
	One pr. IA / 2+ pr. IA	0.69	0.62, 0.76	0.0001
Activity of the mother	Active / housewife	0.87	0.80, 0.94	0.0010
Type of activity of the mother	Manual / non-manual	1.10	1.03, 1.17	0.0024
Education of the mother	Without + technical / secondary + university	1.18	1.11, 1.26	0.0001
Education of the father	Without + technical / secondary + university	1.05	0.99, 1.12	0.1091

[1]1988: first trimester only

age group, as detected previously by the interaction term.

DISCUSSION

The impact of age of the mother on infant mortality has already been examined in numerous papers in the literature, either as an explanatory variable or as a confounding variable. It is well known, for example, that very young mothers or females near the end of their reproductive career have higher risks of fetal and infant mortality (Cramer, 1987; Friede et al., 1988). The present study has shown that older women (35–44 years old) bear children with higher risks of neonatal mortality than children of younger women, though the impact of age remains slighter than that of some characteristics of the reproductive history such as number of previous live births, fetal deaths, and induced abortions. Older mothers also have higher risks of giving birth to children with congenital abnormalities (Naeye, 1983; EUROCAT, 1995).

The importance of age of mother in the study of risk factors for feto-infant survival has concealed to some extent the biological risk factors on the paternal side. Our study shows that the impact of age of father on neonatal mortality is not

TABLE 3

ODDS RATIOS FROM DISCRETE-TIME LOGISTIC REGRESSION MODEL OF POST-NEONATAL MORTALITY AMONG CHILDREN OF MARRIED WOMEN IN HUNGARY 1984–1988[1]

Characteristics	Modalities and Reference Group	OR	95% Confidence Limits	p
Age of mother (in years)	15–24 / 35–44	1.03	0.81, 1.31	0.7753
	25–34 / 35–44	0.87	0.70, 1.08	0.2163
Age of father (in years)	15–24 / 35–44	1.14	0.94, 1.38	0.1849
	25–34 / 35–44	0.99	0.95, 1.16	0.9525
Previous live birth(s) (LB)	No pr. LB / 2+ pr. LB	0.53	0.46, 0.62	0.0001
	One pr. LB / 2+ pr. LB	0.68	0.60, 0.78	0.0001
Previous fetal death(s) (FD)	No pr. FD / 2+ pr. FD	0.67	0.54, 0.83	0.0002
	One pr. FD / 2+ pr. FD	0.77	0.60, 0.98	0.0320
Previous induced abortion(s) (IA)	No pr. IA / 2+ pr. IA	0.72	0.60, 0.88	0.0010
	One pr. IA / 2+ pr. IA	0.79	0.64, 0.99	0.0417
Activity of the mother	Active / housewife	0.67	0.58, 0.76	0.0001
Type of activity of the mother	Manual / non-manual	1.10	0.99, 1.24	0.0671
Education of the mother	Without + technical / secondary + university	1.32	1.17, 1.48	0.0001
Education of the father	Without + technical / secondary + university	1.22	1.09, 1.36	0.0006

[1]1988: first trimester only

TABLE 4

ODDS RATIOS FROM DISCRETE-TIME LOGISTIC REGRESSION MODEL OF NEONATAL MORTALITY AMONG BOYS OF MARRIED WOMEN IN HUNGARY 1984–1988[1]

Characteristics	Modalities and Reference Group	OR	95% Confidence Limits	p
Age of mother (in years)	15–24 / 35–44	0.76	0.65, 0.89	0.0007
	25–34 / 35–44	0.76	0.66, 0.87	0.0001
Age of father (in years)	15–24 / 35–44	0.84	0.73, 0.96	0.0087
	25–34 / 35–44	0.83	0.75, 0.92	0.0004
Previous live birth(s) (LB)	No pr. LB / 2+ pr. LB	0.81	0.73, 0.90	0.0001
	One pr. LB / 2+ pr. LB	0.70	0.64, 0.77	0.0001
Previous fetal death(s) (FD)	No pr. FD / 2+ pr. FD	0.37	0.33, 0.42	0.0001
	One pr. FD / 2+ pr. FD	0.56	0.48, 0.65	0.0001
Previous induced abortion(s) (IA)	No pr. IA / 2+ pr. IA	0.46	0.40, 0.51	0.0001
	One pr. IA / 2+ pr. IA	0.65	0.57, 0.75	0.0001
Activity of the mother	Active / housewife	0.89	0.80, 1.00	0.0440
Type of activity of the mother	Manual / non-manual	1.13	1.04, 1.23	0.0023
Education of the mother	Without + technical / secondary + university	1.18	1.09, 1.29	0.0001
Education of the father	Without + technical / secondary + university	1.06	0.97, 1.15	0.2134

[1]1988: first trimester only

TABLE 5

ODDS RATIOS FROM DISCRETE-TIME LOGISTIC REGRESSION MODEL OF NEONATAL MORTALITY AMONG GIRLS OF MARRIED WOMEN IN HUNGARY 1984–1988*

Characteristic	Modalities and Reference Group	OR	95% Confidence Limits	p
Age of mother (in years)	15–24 / 35–44	0.76	0.63, 0.91	0.0031
	25–34 / 35–44	0.78	0.67, 0.92	0.0026
Age of father (in years)	15–24/ 35–44	0.94	0.81, 1.10	0.4376
	25–34 / 35–44	0.82	0.73, 0.92	0.0012
Previous live birth(s) (LB)	No pr. LB / 2+ pr. LB	0.74	0.66, 0.84	0.0001
	One pr. LB / 2+ pr. LB	0.75	0.68, 0.84	0.0001
Previous fetal death(s) (FD)	No pr. FD / 2+ pr. FD	0.34	0.29, 0.39	0.0001
	One pr. FD / 2+ pr. FD	0.56	0.48, 0.66	0.0001
Previous induced abortion(s) (IA)	No pr. IA / 2+ pr. IA	0.52	0.45, 0.61	0.0001
	One pr. IA / 2+ pr. IA	0.74	0.63, 0.87	0.0004
Activity of the mother	Active / housewife	0.84	0.74, 0.95	0.0064
Type of activity of the mother	Manual / non-manual	1.05	0.96, 1.16	0.2728
Education of the mother	Without + technical / secondary + university	1.18	1.07, 1.31	0.0009
Education of the father	Without + technical / secondary + university	1.05	0.95, 1.16	0.3159

*1988: first semester only

very different from that of age of mother once confounding factors are controlled for. Older fathers in the age group 35–44, compared to fathers aged 25–34, lead to higher neonatal mortality, for both sons and daughters. The absence of a differential for daughters of fathers aged 15–24 could be due to a smaller number of neonatal deaths for females than for males. It should be pointed out, however, that L. E. Gavrilov and N. S. Gavrilova (2001c) have found that daughters of very young fathers (under 25 years) have shorter life spans. They have hypothesized that this result could be "caused by the residual genomic imprinting of the germ cell DNA in particularly young males" (p. 26).

As ten-year age groups are used, we have to be sure that our results on age of father and child mortality are not contaminated by the confounding effect of age of mother. The use of five-year age groups has led to the same results, though with wider confidence intervals, as could be expected. In addition, we have checked our results excluding from the analysis all mothers older than 30 years of age. Our conclusions remain the same; the impact of age of father on child survival at very young ages seems therefore genuine.

The literature shows that age of the father could possibly be linked to some types of dominant autosomal mutations. This would be the case, for example, with achondroplasia, Marfan syndrome, Apert syndrome, osteogenesis imperfecta, and fibrodysplasia ossificans progressiva (Friedman, 1981; Bordson and Sears Leonardo, 1991; Gavrilov and Gavrilova, 2001a), even when controlling for age of mother (Murdoch et al., 1972; Lian et al., 1986; Risch et al., 1987). Moreover, malformations due to structural chromosomal abnormalities are mainly attributed to paternal genetic factors. These types of abnormalities could be responsible for 5 percent of pregnancy losses (Rosenbusch and Sterzik, 1991). Furthermore, numerous studies have analyzed the

quantity and quality of human sperm. While the first studies were mainly concerned with sperm concentration (Carlsen et al., 1992), the following have been focused more on semen characteristics, such as sperm chromosome analysis. Even if the types of studies differ, and even if they were often performed on small numbers of men, results converge: the frequency of spermatozoa with chromosomal abnormalities is around 10 percent, with a majority being due to structural chromosomal abnormalities (6 or 7 percent) (Brandriff et al., 1985; Martin et al., 1987; Guttenbach et al., 1997). Furthermore, an increase in sperm structural abnormalities was significantly correlated with an increasing age in men (Martin and Rademaker, 1987). The frequency increases from 2.8 percent for the youngest age group (20–24 years) to 7.8 percent between 35 and 45 years of age, and it reaches the value of 13.6 percent above age 45. However, other studies (Rosenbusch et al., 1992; Estop et al., 1995) failed to reach a level of statistical significance even if a trend of increasing frequencies was indeed observed at higher ages. Moreover, Spandorfer et al. (1998) have found no differences in the concentration, motility, or morphology of the spermatozoa with paternal aging. One hypothesis for explaining the impact of paternal age on structural abnormalities is errors in DNA transcription during the process of spermatogenesis (Chandley, 1991). As spermatogenesis is a lifelong process, an increase in errors could occur among males at higher ages because of the numerous divisions of the undifferentiated spermatogonia (Bordson and Sears Leonardo, 1991).

These results agree with those relating to the etiology of malformations of paternal origin, but behavioral factors must also not be ignored. Indeed, a decrease in the frequency of sexual intercourse with age leads to an aging of spermatozoa if these stay in the male genital tract over a long period. Aged spermatozoa often present structural abnormalities (Guerrero and Rojas, 1975; Munné and Estop, 1993). It is therefore difficult to distinguish between the effect due to changing behaviors with age from those due to natural aging of the gonads with increasing age. However, as Schwartz et al. (1983) have shown, analysis of the ejaculates obtained after a length of abstinence of 5 days or less show that the frequency of morphologic sperm abnormalities in the older age groups (more than 40 years) is significantly higher than that found in the group 26 to 35.

Biological determinants have been stressed in this paper for two reasons. The first is that the impact of paternal age is greater the younger the child, suggesting a biological explanation. The second is that we have found no other plausible explanations in our review of the literature. Would older fathers take less care of their children than younger fathers, controlling for maternal age? Could possible social causes be associated with father's age? Could socioeconomic status explain some part of the relationship? Unfortunately, our information on socioeconomic status was either scant or difficult to interpret for this period under communism. These questions thus remain open at the present time. Moreover, in order to control for possible cohort effects, longitudinal studies would be required here to study the effect of paternal age.

To conclude, our study shows that not only age of the mother but also the age of the father has a statistically significant impact on the survival of the child during the neonatal period. This relationship holds even when controlling for possible confounding variables. The results discussed in this paper are based on data from a low-mortality country. It would be interesting to enlarge this data set to take account of countries with higher mortality. Concerning the causes of this effect, numerous studies drawn from the biological sphere point to the possible impact of the deteriorating quality of human sperm with increasing age of father. However, what are the respective impacts of the decreasing coital frequency with age, the aging of the genetic material carried by the spermatozoa, and the aging of the spermatozoa themselves in the male genital tract due to lower frequency of intercourse? Should one conclude that "it is good public health policy to recommend that both men and women complete their family before age 40, if possible" (Friedman, 1981, p. 745)?

ACKNOWLEDGMENTS

We would like to thank Dr. Peter Jozan and the Hungarian Statistical Office for making available the individual vital registration data files from the years 1984 to 1988. Many thanks, also, to Dr. Iqbal Shah and Dr. Michael T. Mbizvo (World Health Organization), and to two anonymous referees for valuable comments and suggestions. This research has been initiated in the framework of an INTAS project (Grant no. 93-1617) of the European Community on *The mechanisms of human longevity, an association of parental characteristics and longevity of their children*, in collaboration with Dr. Leonid Gavrilov and Dr. Natalia Gavrilova (Moscow).

REFERENCES

Akoto, E., and D. Tabutin. 1989. Les inégalités socio-économiques et culturelles devant la mort, pp. 35–63. *In* G. Pison, E. van de Walle, M. Sala-Diakanda (eds.), Mortalité et société en Afrique. INED—UIESP—IFORD—MHN, Travaux et Documents, Cahier n° 124. Paris: PUF.

Arntzen, A., P. Magnus, and L. S. Bakketeig. 1993. Different effects of maternal and paternal education on early mortality in Norway. Paediatr. Perinat. Epidemiol. 7:376–386.

Bordson, B. L., and V. Sears Leonardo. 1991. The appropriate upper age limit for semen donors: A review of the genetic effects of paternal age. Fertil. Steril. 56(3):397–401.

Brandriff, B., L. Gordon, L. Ashworth, G. Watchmaker, D. Moore II, A. J. Wyrobek, and A. V. Carrano. 1985. Chromosomes of human sperm: Variability among normal individuals. Hum. Genet. 70:13–24.

Bross, D. S., and S. Shapiro. 1982. Direct and indirect associations of five factors with infant mortality. Am. J. Epidemiol. 115(1):78–91.

Caldwell, J. C. 1979. Education as a factor in mortality decline. An examination of Nigerian data. Pop. Stud. 33(3):395–413.

Carlsen, E., A. Giwercman, N. Keiding, and A. V. Skakkebaek. 1992. Evidence for decreasing quality of semen during past 50 years. Br. Med. J. 305:609–13.

Chandley, A. C. 1991. On the parental origin of de novo mutation in man. J. Med. Genet. 28:217–223.

Cramer, J. C. 1987. Social factors and infant mortality: Identifying high-risk groups and proximate causes. Demography 24(3):299–322.

Demont, F., J. H. Soutoul, F. Pierre, A. Decaux, B. Grandbastien, and M. Gicquel. 1990. Exploration de la législation périnatale en Europe, pp. 85–96. *In* F. Puech and B. Blanc (eds.), 20ème Journées Nationales de Médecine Périnatale. Paris: Arnette.

Estop, A. M., C. Marquez, S. Munné, J. Navarro, K. Cieply, V. Van Kirk, M. R. Martorell, J. Benet, and C. Templado. 1995. An analysis of human sperm chromosome breakpoints. Am. J. Hum. Genet. 56:452–460.

EUROCAT. 1995. Report 6—Surveillance of congenital anomalies in Europe 1980–1992. Brussels: European Registration of Congenital Anomalies Working Group/Institute of Hygiene and Epidemiology.

Friede, A., W. Baldwin, P. Rhodes, J. Buehler, and L. Strauss. 1988. Older maternal age and infant mortality in the United States. Obstet. Gynecol. 72(2):152–157.

Friedman, J. M. 1981. Genetic disease in the offspring of older fathers. Obstet. Gynecol. 57(6):745–749.

Gavrilov, L. A., and N. S. Gavrilova. 2001a. Epidemiology of human longevity: The search for appropriate methodology. J. Anti-Aging Med. 4(1):13–30.

Gavrilov, L. A., and N. S. Gavrilova. 2001b. Biodemographic study of familial determinants of human longevity. Population—An English selection 13(1):197–222.

Gavrilov, L. A., and N. S. Gavrilova. 2001c. Human longevity and parental age at conception, pp. 7–31. *In* J.-M. Robine, T. B. L. Kirkwood, and M. Allard (eds.), Sex and Longevity: Sexuality, Gender, Reproduction, Parenthood. Berlin, Heidelberg: Springer-Verlag.

Gavrilov, L. A., and N. S. Gavrilova. 1997. Parental age at conception and offspring longevity. Rev. Clin. Gerontol. 7:5–12.

Golding, J. 1991. The epidemiology of perinatal death, pp. 401–436. *In* M. Kiely (ed.), Reproductive and Perinatal Epidemiology. Boca Raton, FL: CRC Press.

Gourbin, C. 2002. La mortalité fœtale, pp. 211–249. *In* G. Caselli, J. Vallin, and G. Wunsch (eds.), Démographie : analyse et synthèse, Vol. II. Les déterminants de la fécondité. Paris: Editions de l'INED.

Gourbin, C., and G. Wunsch. 1999. Age differentials in infant mortality. Genus LV(1–2):61–72.

Guerrero, R. V., and O. I. Rojas. 1975. Spontaneous abortion and aging of human ova and spermatozoa. N. Engl. J. Med. 293:573–575.

Guttenbach, M., W. Engel, and M. Schmid. 1997. Analysis of structural and numerical chromosome abnormalities in sperm of normal men and carriers of constitutional chromosome aberrations. A review. Hum. Genet. 100:1–21.

Haines, M. R. 1995. Socio-economic differentials in infant and child mortality during mortality decline: England and Wales, 1890–1911. Pop. Stud. 49(2):297–315.

Leridon, H. 1987. La mortalité foetale spontanée: Rôle de l'âge de la mère, de la parité et des antécédents d'avortements. J. Gynecol. Obstet. Biol. Reprod. 16:425–431.

Lian, Z. H., M. M. Zack, and J. D. Erickson. 1986. Paternal age and the occurrence of birth defects. Am. J. Hum. Genet. 39:648–660.

Macfarlane, A., and M. Mugford. 1984. Birth counts. Statistics of pregnancy and childbirth. London: HMSO Books.

Majumder, A. K., M. May, and P. D. Pant. 1997. Infant and child mortality determinants in Bangladesh: Are they changing? J. Biosoc. Sci. 29(4):385–399.

Martin, R. H., and A. W. Rademaker. 1987. The effect of age on the frequency of sperm chromosomal abnormalities in normal men. Am. J. Hum. Genet. 41:484–492.

Martin, R. H., A. W. Rademaker, K. Hildebrand, L. Long-Simpson, D. Peterson, and J. Yamamoto. 1987. Variation in the frequency and type of sperm chromosomal abnormalities among normal men. Hum. Genet. 77:108–114.

Masuy-Stroobant, G. 2002. Les déterminants de la santé et de la mortalité infantiles, pp. 129–144. *In* G. Caselli, J. Vallin, and G. Wunsch (eds.), Démographie : analyse et synthèse. Vol. III. Les déterminants de la mortalité. Paris: Editions de l'INED.

Masuy-Stroobant, G. 1988. Santé de l'enfant et inégalités sociales. Louvain-la-Neuve: Ciaco.

Menard, S. 1995. Applied logistic regression analysis. Sage University Paper Series on Quantitative Applications in the Social Sciences, 07-106. Thousand Oaks, CA: Sage.

Miller, J. E. 1989. Is the relationship between birth intervals and perinatal mortality spurious? Evidence from Hungary and Sweden. Pop. Stud. 43(3):479–495.

Munné, S., and A. M. Estop. 1993. Chromosome analysis of human spermatozoa stored in vitro. Hum. Reprod. 8:581–586.

Murdoch, J. L., B. A. Walker, and V. A. McKusick. 1972. Parental age effects on the occurrence of new mutations for the Marfan syndrome. Ann. Hum. Genet. 35:331–336.

Naeye, R. L. 1983. Maternal age, obstetric complications, and the outcome of pregnancy. Obstet. Gynecol. 61(2):210–216.

Ressequie, L. J. 1976. Paternal age, stillbirths, and human mutation. Ann. Hum. Genet. 40:213–219.

Rish, A. H., N. S. Weiss, E. A. Clarke, and A. B. Miller. 1988. Risk factors for spontaneous abortion and its recurrence. Am. J. Epidemiol. 128(2):420–430.

Risch, N., E. W. Reich, M. Wishnick, and J. G. McCarthy. 1987. Spontaneous mutation and parental age in humans. Am. J. Hum. Genet. 41:218–248.

Rosenbusch, B., and K. Sterzik. 1991. Sperm chromosomes and habitual abortion. Fertil. Steril. 56(2):370–372.

Rosenbusch, B., E. Strehler, and K. Sterzik. 1992. Cytogenetics of human spermatozoa: Correlations with sperm morphology and age of fertile men. Fertil. Steril. 58(5):1071–1072.

SAS Institute Inc. 1989. SAS/STAT user's guide, version 6, fourth edition, 1. Cary, NC: SAS Institute, Inc.

Saurel-Cubizolles, M. J., and M. Kaminski. 1986. Work in pregnancy: Its evolving relationship with perinatal outcome (A review). Soc. Sci. Med. 22(4):431–442.

Schwartz, D., M. J. Mayaux, A. Spira, M. L. Moscato, P. Jouannet, F. Czyglik, and G. David. 1983. Semen characteristics as a function of age in 833 fertile men. Fertil. Steril. 39(4):530–535.

Selvin, S., and J. Garfinkel. 1976. Paternal age, maternal age and birth order and the risk of a fetal loss. Hum. Biol. 48(1):223–230.

Spandorfer, S. D., O. A. Avrech, L. T. Colombero, G. D. Palermo, and Z. Rosenwaks. 1998. Effect of parental age on fertilization and pregnancy characteristics in couples treated by intracytoplasmic sperm injection. Hum. Reprod. 13:334–338.

Spira, A., B. Ducot, M. L. Guihard-Moscato, N. Job-Spira, M. J. Mayaux, J. Ménétrier, and J. Wattiaux. 1993. Conception probability and pregnancy outcome in relation to age, cycle regularity and timing of intercourse, pp. 271–284. *In* R. Gray, H. Leridon, and A. Spira (eds.), Biomedical and Demographic Determinants of Reproduction. Oxford: Clarendon Press.

Trussell, J., and C. Hammerslough. 1983. A hazard-model analysis of the covariates of infant and child mortality in Sri-Lanka. Demography 20:1–26.

Tzoumaka-Bakoula, C., V. Lekea-Karanika, N. S. Matsaniotis, and J. Golding. 1989. The Greek National Perinatal Survey. II. Socioeconomic factors and perinatal mortality in Greece. Paediatr. Perinat. Epidemiol. 3(1):43–51.

W.H.O. 1970. The prevention of perinatal mortality and morbidity. Techn. Rep. Ser. 457. Geneva: W.H.O.

W.H.O. 1979. Gestation, birthweight, and spontaneous abortion in pregnancy after induced abortion. Lancet: January 20, 8108(1):142–145.

Fertility and Post-Reproductive Longevity

Ken R. Smith[a,d], Geraldine P. Mineau[b,d], and Lee L. Bean[c]

[a]Department of Family and Consumer Studies, 225 South 1400 East, Rm 228, University of Utah, Salt Lake City, Utah 84112, ken.smith@fcs.utah.edu; [b]Department of Oncological Sciences, 2000 Circle of Hope, University of Utah, Salt Lake City, Utah 84112-0900, geri.mineau@hci.utah.edu; [c]Department of Sociology, 301 Social Behavioral Science Building, University of Utah, Salt Lake City, Utah 84112, bean@freud.sbs.utah.edu; [d]Population Sciences, Huntsman Cancer Institute, 2000 Circle of Hope, University of Utah, Salt Lake City, Utah 84112-0900

Abstract: **We examine the effects of reproduction on longevity among mothers and fathers after age 60. This study is motivated by evolutionary theories of aging and theories predicting social benefits and costs of children to older parents. We use the Utah Population Database, that includes a large genealogical database from the Utah Family History Library. Cox proportional hazard models based on 13,987 couples married between 1860–1899 indicate that women with fewer children as well as those bearing children late in life live longer post-reproductive lives. As the burdens of motherhood increase, the relative gains in longevity of late fertile women increase compared to their non-late fertile counterparts. Husbands' longevity is less sensitive to reproductive history, although husbands have effects that are similar to those of their wives during the latter marriage cohort. We find some support for predictions based on evolutionary principles, but we also find evidence that implicates a role for shared marital environments.**

INTRODUCTION

Childbearing and child rearing affect the lives of parents. It is well known that childbirth has significant health effects on mothers during their childbearing years (National Research Council, 1989). While numerous studies demonstrate the effects of female reproductive history on cancer and heart disease mortality, far less is known about the influences of fertility patterns on the longevity of mothers and fathers who survive their reproductive years. Interest in this question has increased recently as a result of work by evolutionary biologists and biodemographers (Westendorf and Kirkwood, 1998; Rose, 1997; Vaupel et al., 1998). In describing the field of biodemography and its links to evolutionary concepts, Vaupel et al., (1998) recently noted that "It is reproductive success that is optimized [through natural selection], not longevity. Deeper understanding of survival at older ages thus hinges on intensified research into the interactions between fertility and longevity." We suggest, therefore, that it is important for demographers to examine the association between fertility history and post-reproductive aging, particularly from the perspective of both evolutionary and social theories (Wachter and Finch, 1997).

We revisit the issue of fertility and post-reproductive longevity in light of two recent developments. First, evolutionary biologists and biodemographers have recently argued that female fertility patterns in various species, including humans, may be useful markers for rates of biological aging (Kirkwood and Rose, 1991; Vaupel et al., 1998). A reexamination of the association between human female fertility and longevity is therefore warranted based on evolutionary theories of aging. Second, family demographers