Twinning and the Inheritance of High Fecundity

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Draft: 2 April 2008

Paper prepared for the Population Association of America, New Orleans, 16-18 April 2008.

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Scientific interest in historical data to study the heritability of human fertility goes back at least to the work of Karl Pearson (Pearson, Lee et al. 1899), who used the pedigrees of noble and landed families. Evidence drawn from the period before the transition to low fertility is particularly attractive for this purpose, because it seems to offer a way to separate genetic from environmental and behavioral influences. The assumption is that before the spread of voluntary family limitation, genetic influences should be the main source of any correlation in fertility across generations. Unfortunately, the problem turns out to be much more complicated. Even in the absence of birth control within marriage, fertility is affected by ages at marriage, breastfeeding practices, and infant mortality, which have environmental and behavioral origins shared across generations. The central problem is that we observe fertility, a woman's production of live births, and not fecundity, the biological capacity to reproduce. In this paper we take a different strategy by starting with an indicator of a mother's fecundity and looking for its correlation with the fertility of her daughters. We find that high fecundity was inherited by daughters from their mothers and resulted in high fertility in the second generation. This pattern disappears with the transition to low fertility, however, confirming the intuition that the genetic inheritance of fecundity should have more effect on fertility before the spread of fertility control.

Murphy (1999) has reviewed studies of the heritability of fertility, and he finds that the correlation between the family sizes of parents and children are generally very weak, especially among historical studies. Langford and Wilson's (1985) analysis of English family reconstitutions collected by the Cambridge Group concludes that "there is no connection between mothers and daughters where fecundity is concerned" (p. 441). Similarly, Bocquet-Appel and Jakobi (1993) find: "At Arthez d'Asson before the transition, the fertility and nuptiality of a couple were not influenced by their parents, but by their generation" (p. 346). Desjardins et al. (1991) find "hints" of an association between the marriage-first birth intervals of mothers and daughters, but it is very weak (p. 53).

Murphy also notes a tendency for the heritability of fertility to increase over time, as fertility has become more subject to conscious choice. While most research on fertility emphasizes the role of social factors in transmitting family size norms across generations, Udry (1996), Kohler, Rodgers, and Christensen (1999), and Rodgers, Bard, and Miller (2007) argue that biosocial factors play an increasing role in fertility over time.

This paper asks whether we can detect shorter than average birth intervals among the daughters and granddaughters of women with a marker for high fecundity: twinning. Previous studies have concluded that the propensity to have dizygotic twins is genetically related to high fecundity (Bortolus, Parazzini et al. 1999). Women who have twins get pregnant more quickly (Pison and Couvert 2004), and twinning is an inherited trait (Parisi, Gatti et al. 1983). Thus, we can use a family history of twinning to distinguish fecundity, which is a biological potential, from achieved fertility, which is affected by choices like age at marriage and family limitation. We will see that twinning is affected by age and breastfeeding, but these patterns can be traced to underlying biological mechanisms.

Data are drawn from family reconstitutions of eighteenth-century French villages collected by Louis Henry (Henry 1972; Henry and Houdaille 1973; Houdaille 1976; Henry 1978; Séguy, Colençon et al. 2001). These data include the first stage of fertility decline in France, allowing us to ask whether the inheritance of high fecundity became more or less important after the onset of family limitation.

The Epidemiology of Twinning

Most studies find that monozygotic twinning occurs at a constant rate (Bortolus, Parazzini et al. 1999), but there is a rich literature describing the factors affecting dizygotic twinning. Dizygotic twinning has been linked to a woman's level of follicle-stimulating hormone (FSH), which plays a critical role in the complex sequence of events during the ovarian cycle (Scheele and Schoemaker 1996). In each cycle, a cohort of follicles begins to develop in the ovaries. FSH is required during the first stage of follicle growth, and ovulation will not occur if the level of FSH is not sufficient. Most of the time, a dominant follicle emerges, which will mature and extrude an egg during ovulation. When the dominant follicle reaches a certain stage of development, it releases inhibiting factors that suppress FSH, which stops the growth of other follicles (Baird 1987). Multiple births tend to occur when the level of FSH is high, because more than one follicle crosses the developmental threshold leading to ovulation.

Levels of FSH in the bloodstream steadily increase with age, resulting in a rise in the frequency of multiple ovulations. However, loss of ovarian function and the mortality of fertilized ova and embryos also increase with age and become common as menopause approaches. After age 36 or 37, they counteract the increase in multiple ovulations, which explains the drop in the rate of fraternal twins beyond that age. The interactions between follicular growth and FSH change as the number of follicles available for each ovarian cycle decreases (Macklon and Fauser 1999).[1] At older ages, a single follicle might not be sufficiently developed to signal the pituitary system to reduce production of FSH, and two follicles may mature to ovulation in this environment (Macklon and Fauser 1998; Beemsterboer, Homburg et al. 2006; Hoekstra, Zhao et al. 2008). Consequently, the level of FSH tends to rise with age, and a high level of FSH is a precursor of menopause, when the supply of follicles has been exhausted (Djahanbakhch, Ezzati et al. 2007). This implies that inherited differences in FSH production will be less important among younger women, who have a larger pool of growing follicles than among older women, and that differences in fertility related to FSH production should be more pronounced among older than among younger women.

Breastfeeding also has an impact on FSH levels and on twinning. Suckling triggers a neurohormonal response that suppresses the production of FSH (Taya and Sasamoto 1991). Low levels of FSH prevent follicles from maturing into eggs and reduce the

[1] It has been widely assumed that a woman's supply of follicles is created before birth and diminishes with age, but this view has been questioned recently (Djahanbakhch, Ezzati et al. 2007).

production of estrogen, which results in amenorrhea. When breastfeeding ceases, FSH rises and ovulation and menstruation resume. It has also been hypothesized that FSH levels tend to overshoot normal levels during recovery from amenorrhea (Lambalk and Schoemaker 1997).

Nutrition may also play a role in the regulation of FSH and twinning, especially during breastfeeding (Bortolus, Parazzini et al. 1999). Smith and Grove (2002) describe mechanisms linking negative energy balance during lactation to the release of GnRH, which is a regulator of FSH. This points to a link between nutrition and lactation, which has been viewed very differently by demographers and physiologists (Wood 1994). Physiologists have documented a link between nutrition during lactation and amenorrhea (Lunn, Diggory et al. 1988), but demographic studies tend to show that nutrition only affects fertility in cases of extreme malnutrition (Bongaarts 1980). We hope to use twinning rates to investigate the effect of nutrition on fertility in our future research.

We propose Figure 1 as a way of visualizing the effects of variations in FSH production on fertility and twinning. Curve A represents the distribution of hereditary differences in FSH level, which we have shown as a normal curve. Line B indicates the risk of a singleton ovulation, and Curve C shows the risk of a multiple ovulation. At a given age, women who have relatively higher levels of FSH are more likely to ovulate and more likely to have twin births. These twin-prone women are located in the right-hand tail of the distribution. As women age, FSH tends to increase for all women, which moves the distribution of women (Curve A) to the right and increases the proportion of twin births. Even though FSH rises with age, fertility decreases, because the pool of follicles at risk of ovulation decreases and the risk of miscarriage increases.

Figure 1 also implies that breastfeeding will reduce the twinning rate. Since lactation reduces FSH levels, it moves the distribution of women by FSH (Curve A) to the left. This reduces the risk of all births, but it reduces the risk of twin births more than the risk of singleton births. The twin-prone women on the right side of the distribution will tend to return to ovulation sooner than those whose normal level of FSH is lower, and they will have shorter birth intervals. Even these twin-prone women will tend to have singleton births, however, because their FSH level is lower than normal. The percentage of twin births will tend to rise after weaning as the effects of breastfeeding diminish. When breastfeeding ends suddenly because of an infant death, the distribution shifts back to the right, which means that the twinning rate should be higher after an infant death.

Since women who gave birth to a twin are disproportionately drawn from the high-FSH side of the distribution shown in Figure 1, we expect that their daughters and granddaughters will exhibit the characteristics of high FSH levels, regardless of whether they have a twin birth themselves. This implies that women whose mothers/grandmothers had a twin birth will have higher fertility than women whose mothers/grandmothers had only singleton births. Furthermore, differences in fertility due to the inheritance of twin-related fecundity will be greater at older ages, when the supply of follicles is low, than at younger ages.

Data

Data for this study are drawn from the "Enquête de Louis Henry" on the population of France from 1670 to 1829. (See Séguy 2001 for a bibliography.) Henry and his colleagues collected parish register information (baptisms, burials, and marriages) from a sample of villages across France. We use here the "nominative" part of the database, which includes the names used to reconstitute families in 40 parishes. The nominative sample consists of 65,654 families and 182,983 births. Data for 34,812 couples with known dates of marriage ("MF") are published on a CD-ROM included with Séguy et al. (2001). We are grateful to Alain Blum for providing us the data for families that are not linked to a marriage ("fiche E").

To study the inheritance of fertility, we have linked the fertility histories of women who remained in their parish of birth to their mothers' histories. The database consists of two files: a "*parents*" table including information about husband and wife and the duration of their union, and an "*enfants*" table describing the children born to each couple. Although children in the "*enfants*" table are linked to the marriage of their parents, they are not linked to their own marriages in the "*parents*" table. We created these links using information on names, dates of birth, and dates of marriages, which are included in both data tables. Matching names is facilitated by a coding system used by Henry to capture variations in the spelling of names in different documents. We were able to link 9,503 married women to their mothers' marriages. These links also yielded 3,242 cases in which we can link a married woman to her mother's mother's marriage and 2,940 links to a father's mother's marriage. We have also identified 2,710 twin births and 35 triplets in the database. Since we can only link generations when both mother and daughter lived in the same village, the families available for studying inheritance of fertility are the most geographically stable subsample of the population.

Gutierrez and Houdaille (1983) have described twinning rates in these data, and they show large regional and temporal variations. Our preliminary explorations of the data suggest that some of these differences in recording may explain some of these patterns. It is important to remember that the data are derived from records of baptisms, not births. Infants who died before being baptized are not included, and Henry estimates that about three percent of births do not appear in the baptisms. Under-registration may be larger for twins, who had much higher mortality than singleton births. The level of under-registration may also be affected by the availability of priests and by interpretations of Catholic doctrines regarding emergency baptism. The delay between birth and baptism was likely to be greater among families living in sparsely settled areas and mountainous regions, where travel to a chapel for the baptism was difficult. Catholic doctrine does allow for a lay person, such as a midwife, to perform a baptism when a child was in danger of dying before a priest could perform the ritual. This procedure (*ondoiement* in French) is recorded in the database, and there are substantial variations among parishes in the prevalence of this practice. Nevertheless, under-registration of twins should not affect the analysis described here.

The analysis of birth intervals presented below uses only a fraction of the total database. The central problem in data of this kind is unobserved censoring. We only know if a couple was present in the parish and under observation if they experienced an event – a baptism, burial, or marriage. Since we do not know when migrants left the parish, we have incomplete information about time at risk. Henry devised the rules of family reconstitution to make it possible calculate fertility rates from family reconstitutions, and we follow his logic in selecting families for our analysis. To include a couple in our analysis, we must know: the date of marriage, the wife's age or date of birth, exact birthdates (day, month, year) for all children, and an unbiased event that ends observation (usually the death of husband or wife). This reduces the data available for analysis to 16,473 couples and 66,440 births. Since each type of analysis has its own specific requirements, we cannot use all of these observations in every table shown below.

Figure 2 presents movements in fertility in the sample used for analysis. We show Total Marital Fertility Rates calculated by decade for ages 15 to 49 (TMFR) and also for ages 20 to 49 (TMFR20+), because age-specific fertility rates at ages 15 to 19 are often unstable. The calculations in Figure 2 are arranged by time of exposure not by marriage cohort, which is often used in family reconstitution studies. The onset of the fertility transition is very apparent in Figure 2. Before 1780, the Total Marital Fertility Rate varied between 9.2 and 9.7 with no apparent trend. It dropped to 8.9 in the 1790s and continued downward to 6.5 in the 1820s. TMFR20 moved in a narrower range, 7.9 to 8.2, and one might see the value of 7.7 in the 1780s as the first sign of falling fertility.

Age-specific marital fertility rates before and after 1780 are presented in Figure 3. These show the characteristic pattern of larger proportional declines at older ages that Henry associated with the beginning of family limitation. Thus, the size of the percentage decrease in age-specific rates from 1670-1779 to 1780-1829 increases with age from a 4 percent decrease at ages 20 to 24 to a 47 percent drop at ages 45 to 49 (see Table 1).

Twinning and Fertility

As Figure 4 shows, women who gave birth to twins had higher fertility at all ages than those who had only single births. The TMFR of women with at least one twin birth was 11.1 children (counting twin births as only one child), compared to 8.9 for women with only singleton births. While this shows that women with twins had higher fertility, it does not show a biological relationship between twinning and fertility. The difference could be biological, in the sense that women with a propensity to have twins could have higher fecundity, or it could be purely statistical, because women with higher fertility have more opportunities to have a twin birth.

We examine this issue in Figures 5 and 6 by examining the time between marriage and first birth for twin and singleton births. In Figure 5 we show the proportion of twin births by time since marriage. Twins were much more common among first births that occurred less than 260 days after marriage. This is partly due to a higher proportion of twins among women who conceived before marriage, which implies that women with a propensity to give birth to twins became pregnant more easily than other unmarried

women (Pison and Couvert 2004). Twins were also unusually common among births 240 to 260 days after marriage. Most of these births were conceived after marriage, but twin births are often premature (the duration of twin pregnancies is on average 14 days shorter than singleton ones's in industrialized countries nowadays). The result, depicted in Figure 6, is that women who had twins completed the first birth interval more rapidly than other women. The median interval between marriage and first birth was 345 days for twins and 380 days for singleton births. In Table 2 we verify these results using logistic regression to be sure that they are not due to differences in age at marriage or time period. In short, the 77 day difference between the average time between marriage and first birth for twin and singleton births results from the combination of two phenomena: mothers of twins become pregnant more rapidly than other women, and twin pregnancies are shorter than singleton ones.

We now turn to the intervals between successive births. The model illustrated in Figure 1 suggests that the relationship between twinning and fertility can be complicated by breastfeeding. Most women breastfed their infants in pre-industrial France for a long time, and lactation increased birth intervals by delaying the return of ovulation. We can observe the effect of breastfeeding by comparing women with a living infant to women who ended breastfeeding early because their child died. In our subsample of the Henry data, the average completed birth interval following a child that survived for at least nine months was 2.59 years, but if the previous child died in less than nine months, the average birth interval was only 1.79 years. We use the survival of the previous child to help us understand the relationship between twinning and fecundity.

If we simply compared birth intervals ending in a twin to those ending with a singleton, we would mistakenly infer that twinning was associated with lower fecundity. The average birth interval ending in a twin birth was slightly longer (2.52 years) than the intervals before singleton births (2.46 years) in pre-industrial France. We argued above, however, that women who are prone to twin births may be more likely to have singleton births when their fecundity is reduced by lactation. This is what we see in the Henry data. Among women whose infants survived for at least nine months, most of whom were breastfeeding, the average birth interval was 2.61 years for women who only gave birth to singletons, but the average birth interval for women who gave birth to at least one twin was 2.38 years for singleton births and 2.73 years for twin births (Table 3). If the previous child died in less than nine months, the shortest average birth intervals were those ending in a twin birth (1.67 years), followed by singleton births to mothers of twins (1.75 years), and the longest intervals were those of women who only had singletons (1.80 years). Thus, women who ever gave birth to a twin always had shorter average birth intervals than women who only had singletons, but breastfeeding increased the likelihood that the next birth would be a singleton among twin-prone women.

In short, our analysis of birth intervals in eighteenth-century France confirms the expectation that women who had twins have higher fecundity than other women. Detecting this difference among married women is complicated by the effects of breastfeeding, but the results match the predictions of our model of the effects of FSH in

Figure 1. We now turn to a model for detecting the effect of inheritance of high fecundity on fertility.

A Model for Birth Intervals

We use Cox (1972) partial likelihood regression to determine whether women who gave birth to twins passed higher fertility to their daughters. The Cox model assumes that the transition process for an event can be described by

$$h(t) = h_0(t)e^{\beta x}$$

in which $h(t)$ is the instantaneous rate of transition (hazard rate) at time t, $h_0(t)$ is the hazard rate for some arbitrary standard individual, x is a vector of covariates, and β is a vector of coefficients. In this application, the transition is giving birth to a child, and time is measured from the birth of the previous child. We present our results in the form of relative risks, which are obtained by exponentiating the estimated coefficients. A relative risk of 2.0 implies that a one unit change in that covariate doubles the hazard of a birth, while a relative risk of 0.5 implies that a one unit change reduces the hazard by 50 percent. If the relative risk is 1.0, the covariate has no effect on the transition rate.

In Table 4, we present models for the inheritance of fertility from mother to daughter. Only women with at least one birth are included. The model includes the following control variables:

Age. The relative risks of a birth vary by age in the expected way, and this pattern is stable across subsamples of the data .

Married less than five years: Relative risks of a birth were 30 percent higher in the first five years of marriage.

Previous infant alive: This time-varying covariate is used to capture the effects of breastfeeding. It is set to one after the birth of a child and to zero when the child reaches 21 months, which assumes twelve months of breastfeeding plus nine months for gestation. If the previous child dies before reaching 12 months, we set this variable to zero nine months after the child's death. For twin births, we use the death of the child that lived the longest. Table 4 shows that breastfeeding has a very powerful effect. The presence of an infant reduced the risk of a birth by about 50 percent in this population.

Region: We find substantial differences in fertility among regions in France, especially before 1780, when fertility was a third lower in the Southwest than in the Northeast.

Quarter century: These binary variables for 25-year intervals show the same trend in fertility as Figure 1. There is no trend before 1775 and a steady decline after 1775.

Inheritance of High Fecundity

We have constructed a variable to identify inheritance of high fecundity by identifying whether a woman's mother had at least one twin birth. When we use this variable, the sample is restricted to women who were linked to their mother. Women who could not be linked across generations are excluded from analysis when this covariate is used. In Table 4 we find higher fertility among women whose mothers had a twin birth. The relative risk of a birth was 5 percent higher (1.05) among women whose mothers had a twin than among women whose mothers had only singleton births. Model 2 in Table 4 asks whether the effect of inheritance changed over time by adding an interaction between the twinning covariate and period. Since the interactions are all comparisons to the omitted category (mother had only singletons between 1750 and 1774), we have computed period-specific effects in the final column of the table. These period-specific effects are obtained by multiplying the relative risk for the interaction effect by the relative risk for the main effect of the inheritance covariate. Thus, they show the effect of inheritance in each period, but they do not include the main period effects. For example, the period-specific effect of having a mother who had twins is 1.08 (=1.02 x 1.06) for women observed from 1675 to 1699. The period-specific effects suggest that twinning in the mother's generation became less predictive of higher fertility in the daughter's generation over time. Before 1775, all of the net relative risks are above 1.0 with a high of 1.22 in 1700-1725. The two estimates after 1775 are 0.96 and 1.00, which show no positive effect on fertility from having a mother who had twins.

The estimates in Table 4 are computed on all the available data in the Henry family reconstitutions without any form of weighting. There were substantial differences among regions, however, which could affect these results. As we noted above, the level of fertility in Southwest France was a third lower than the level in the Northeast. Figure 7 compares trends in the Total Marital Fertility Rate above age 20 across regions. Differences in both behavior (e.g. breastfeeding), recording (e.g. the use of *ondoiement*), and changes in the availability of data by regions can affect both estimates of fertility and twinning across regions.[2] For this reason, Table 5 presents models of fertility separately by region.

The strongest effects of inheritance on fertility appear in the Northeast and Southeast. In both of these regions the relative risk of a birth was higher among women whose mothers had a twin. But there is also a strong interaction between inheritance and period in both regions. In the Northeast, the inheritance effect is strong (relative risk as high as 1.41) before 1775, but it disappears after 1775.[3] In the Southeast, the highest net effects of inheritance are in the two earliest periods (1.98 for 1675-1699; 1.56 for 1700-1725). The estimates after 1725 in the Southeast are lower but still above 1.0. We find no

[2] The twinning rate in the Southwest (1.12 percent) was well below the other regions (Northeast 1.65, Northwest 1.78, Southeast 1.49).

[3] The difference between the interaction effects for 1775-1799 and 1750-1774 (the reference period) is statistically significant.

relationship between inheritance and fertility in the Northwest, and in the Southwest the effect appears to be negative except for 1700 to 1724.

If fecundity is inherited, the strength of this relation in the models estimated here will be affected by both the effectiveness of twinning as an indicator of high fecundity in the mother's generation and the correlation between fecundity and fertility in the daughter's generation. As we showed above, breastfeeding and infant mortality both affect the twinning rate, and underregistration of early infant deaths will have an impact on the recording of twins, who had much higher infant mortality than singletons. We suspect that regional differences in the inheritance effect are related to differences in the quality of twinning as an indicator of high fecundity.[4] This is most likely in the Southwest, which had lower recorded fertility, lower twinning rates, and higher estimated underregistration of births (Henry 1978). The absence of the inheritance effect after 1775, however, seems to be due to changes in behavior in the daughters' generation. We are impressed by the coincidence between the disappearance of the inheritance effect and the onset of the transition to low fertility, especially in the Northeast. Differences in fecundity should be less important in a population that is controlling its fertility, because more fecund women will be more highly motivated to use birth control. We are continuing to search for a test that will allow us to test this hypothesis directly.

Conclusion

Biomedical research suggests that women who have (dizygotic) twins tend to have higher levels of hormones making them more likely to conceive than other women. We have used this relationship to look for the transmission of high fecundity across generations. We began by asking whether birth intervals resulting in twins are shorter than birth intervals for single births, and then we asked whether the daughters of women who had twins also had shorter than average birth intervals. Our analysis of the timing of first births showed that twins were unusually common among premarital conceptions and among births conceived in the first few days after marriage. Both of these suggest that mothers of twins became pregnant more rapidly than other women. We also showed that twin-prone women had shorter birth intervals than women who only gave birth to singletons, but breastfeeding reduced the likelihood that a twin-prone woman would have a twin birth. These results imply that differences in breastfeeding and infant mortality have an impact on the twinning rate, which may explain differences between studies that have attempted to show the inheritance of twinning.

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[4] Monozygotic twinning, which is due to a different process than dizygotic twinning, may not be correlated with fecundity. Since we cannot identify which same-sex twins were monozygotic, mother's twinning should be a less reliable predictor of high fertility when the proportion of monozygotic twins is higher. Thus, a lower twinning rate implies a lower signal to noise ratio in our indicator of high fecundity.

We found evidence that high fecundity (indicated by a mother who had a twin birth) was inherited, but the strength of this relationship varied by period and by region. When all of the Henry data is pooled, the relative risk of a birth was 6 percent higher among women whose mothers had twins than among other women, but this effect is not present after 1775. We also found large regional differences in the inheritance effect, which seem to be due to differences in behavior and data quality. The region with the clearest evidence of inheritance of fecundity, the Northeast, also has the sharpest decrease in this effect after 1775. Since fertility rates were falling after 1780, it seems reasonable to hypothesize that biological differences in fertility are hidden by the practice of family limitation.

Figure 1. Illustrative Model of FSH and Twinning

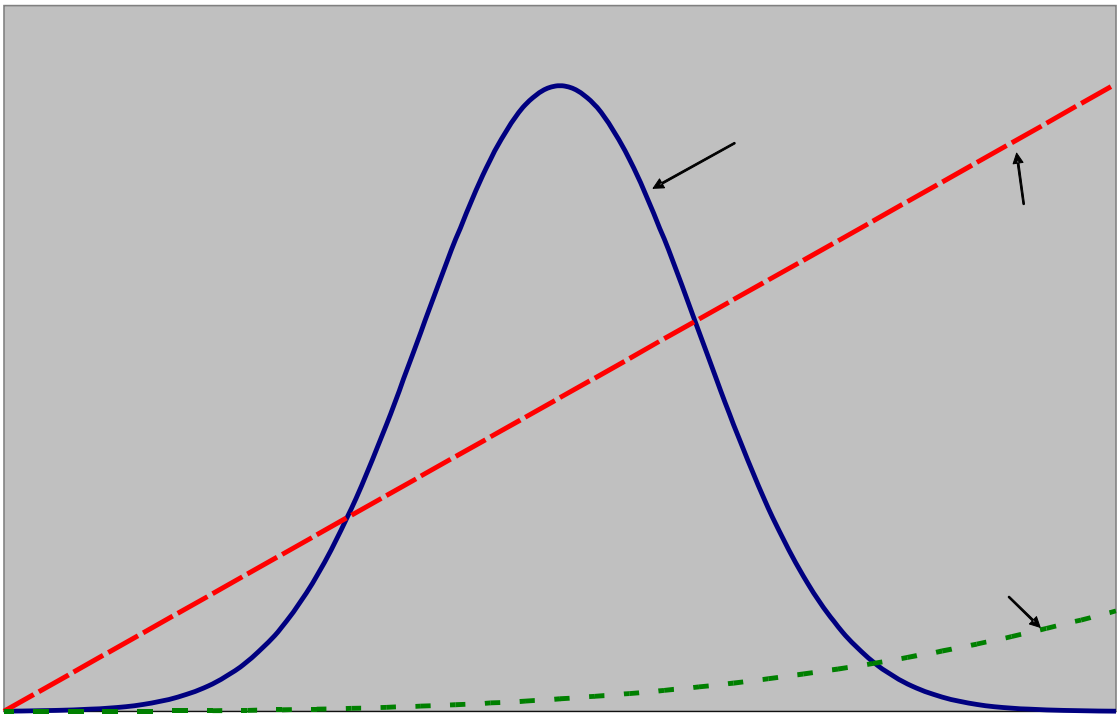


Figure 2. Total Marital Fertility for Ages 15-49 (TMFR) and 20-49 (TMFR20), Henry Family Reconstitutions, France, 1670-1829

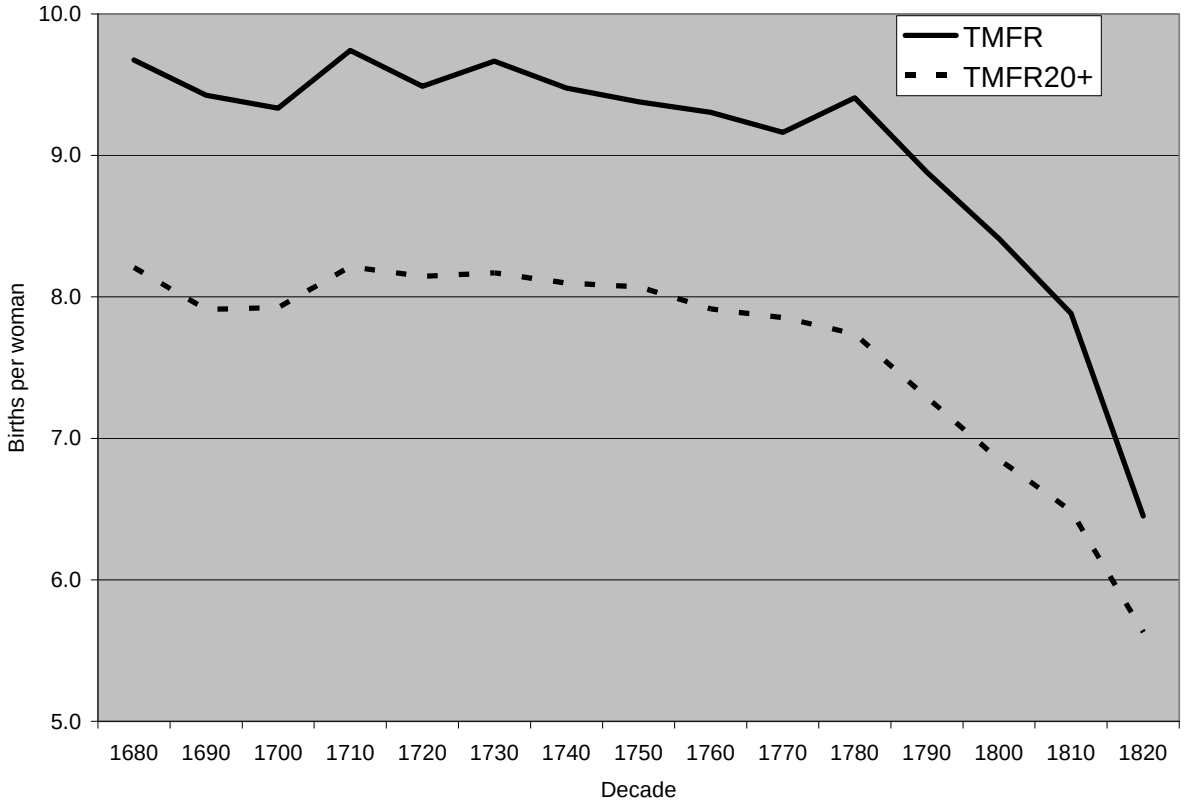


Figure 3. Age-specific Marital Fertility Rates Before and After 1780, Henry Family Reconstitutions, France, 1670-1829

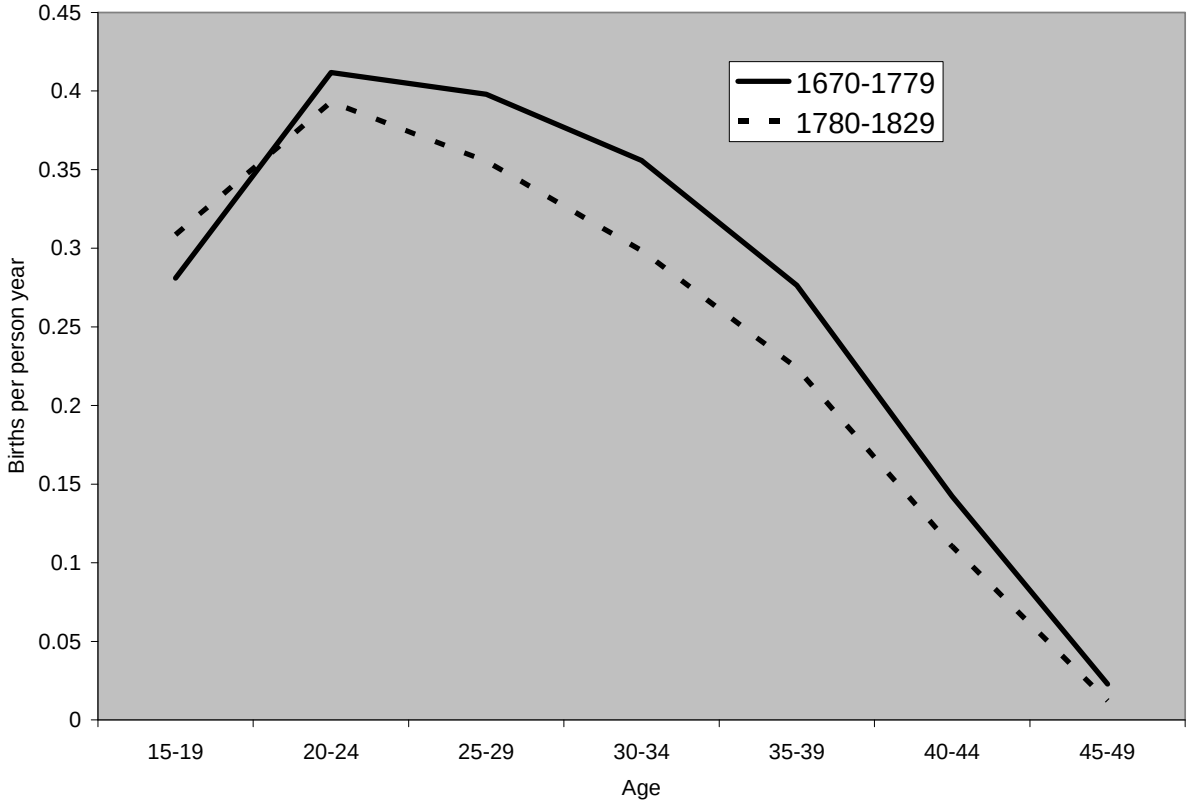
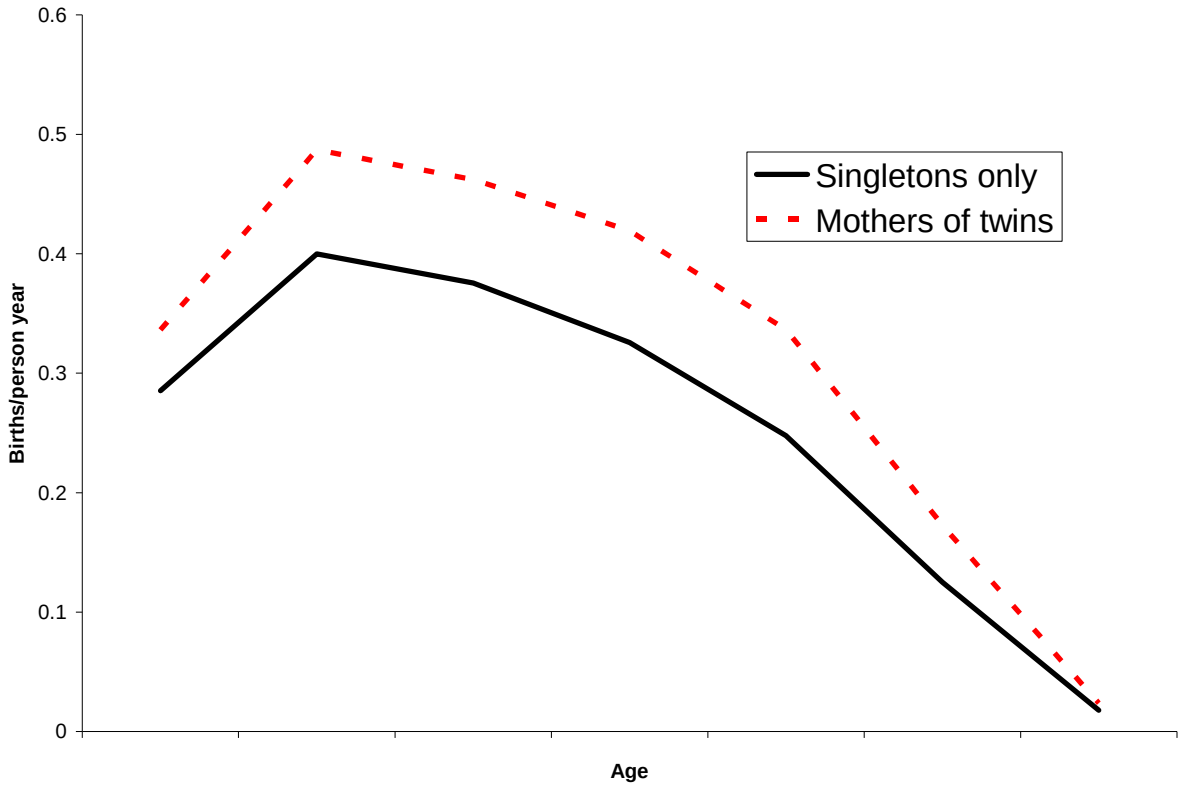


Figure 4. Age-specific Marital Fertility Rates* for Mothers of Singletons and Twins, Henry Family Reconstitutions, France, 1670-1829



* Twin births are counted as only one child in this calculation.

Figure 5. Percent of Twins Among First Births by Time Since Marriage, Henry Family Reconstitutions, France, 1670-1829

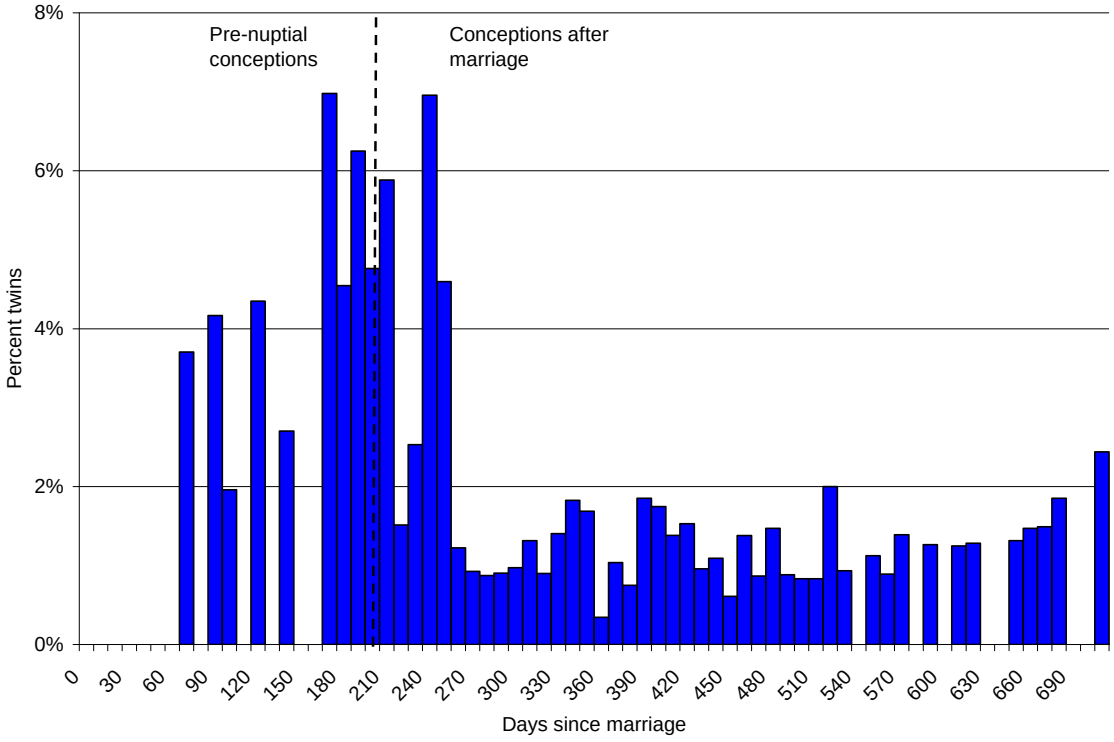


Figure 6. Percent of First Births by Days since Marriage for Singletons and Twins, Henry Family Reconstitutions, France, 1670-1829

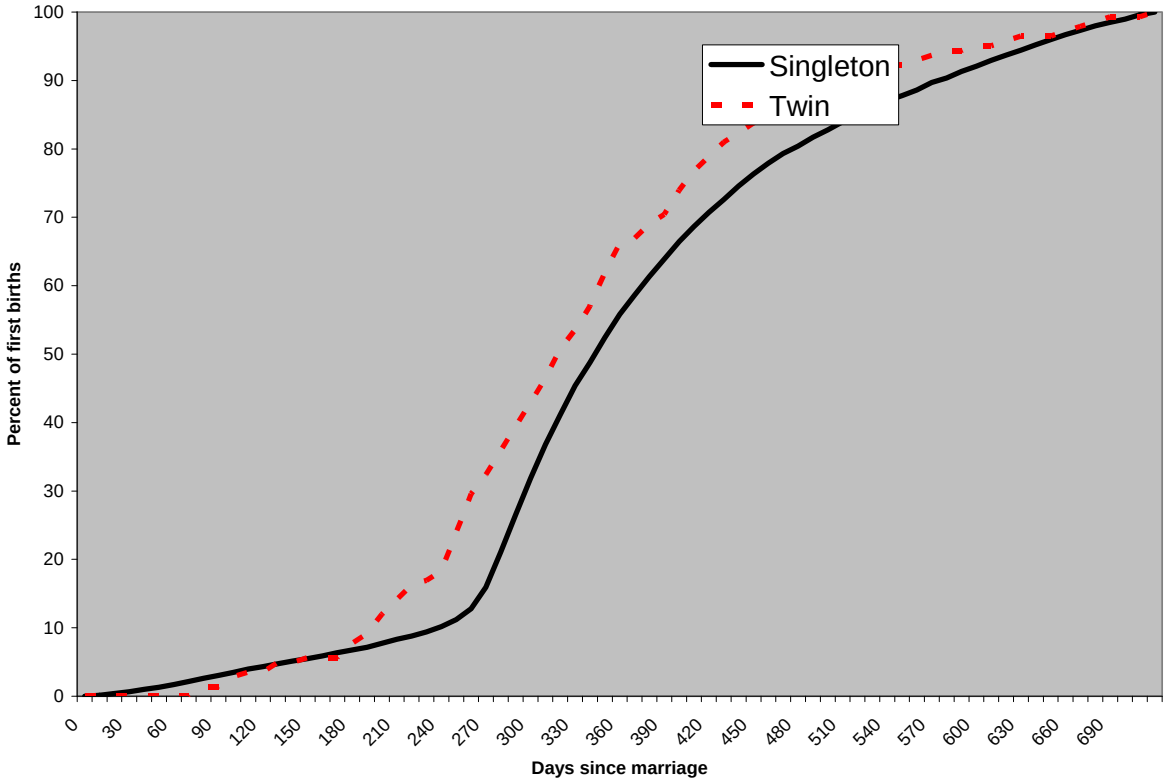


Figure 7. Total Marital Fertility Ages 20 and Older by Period and Region, Henry Family Reconstitutions, Frand, 1680-1829

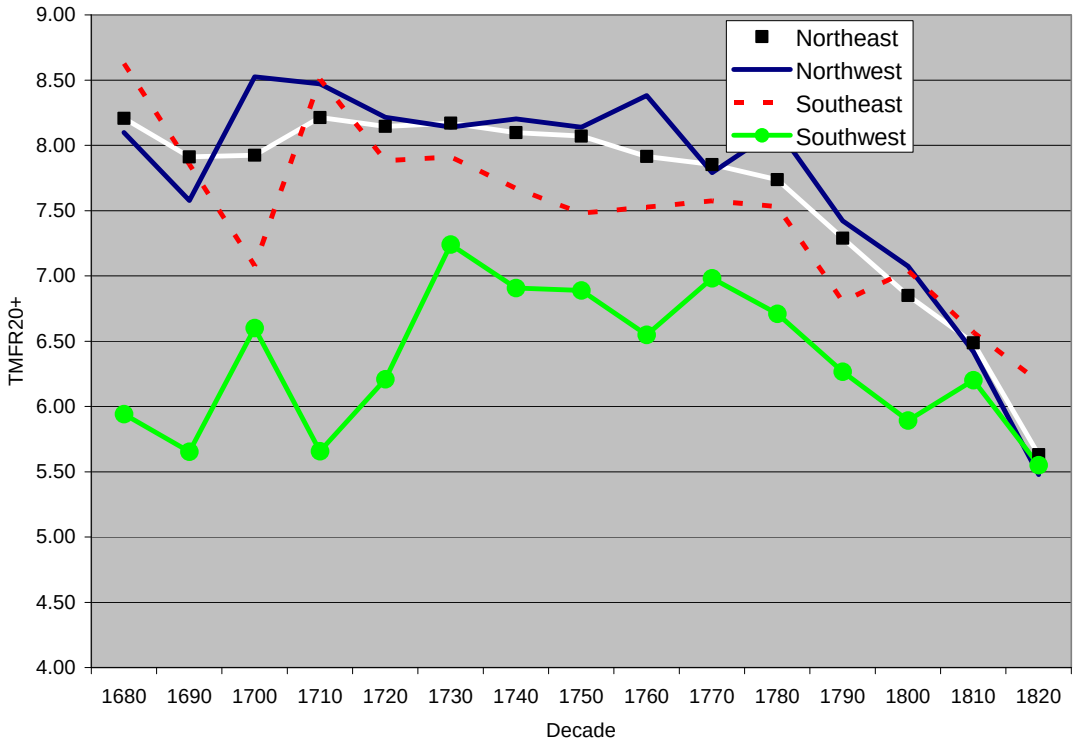


Table 1. Age-specific Marital Fertility Rates Before and After 1780, Henry Family Reconstitutions, France, 1670-1829

	1670-1779	1780-1829	Percent decrease
15-19	0.281	0.309	10%
20-24	0.412	0.393	-4%
25-29	0.398	0.355	-11%
30-34	0.356	0.298	-16%
35-39	0.276	0.224	-19%
40-44	0.142	0.110	-22%
45-49	0.023	0.012	-47%
TMFR	9.4	8.5	-10%

Table 2. Logistic Regression on the Probability of Twins on the First Birth, Henry Family Reconstitutions, France, 1670-1829

Covariate	Odds Ratio	S.E.	
Days between marriage and birth:			
1-239	2.15	0.79	*
240-279	1.75	0.64	
280-299	0.84	0.36	
300-319	1.00		
320-449	1.12	0.38	
450-599	0.89	0.35	
600+	1.07	0.37	
Mother's age:			
15-19	0.43	0.13	**
20-24	0.48	0.09	**
25-29	1.00		
30-34	1.12	0.25	
35-39	1.64	0.49	
40-44	1.21	0.88	
Region:			
Northeast	1.00		
Northwest	0.99	0.19	
Southeast	0.71	0.15	
Southwest	0.47	0.14	*
Decade:			
1670s & 80s	0.63	0.26	
1690s	1.50	0.54	
1700s	0.50	0.24	
1710s	0.59	0.25	
1720s	0.61	0.24	
1730s	1.05	0.36	
1740s	0.92	0.31	
1750s	1.00		
1760s	0.59	0.22	
1770s	0.43	0.18	*
1780s	0.44	0.18	*
1790s	0.72	0.25	
1800s	0.90	0.29	
1810s	0.46	0.18	*
Observations	12882		
Chi-squared	80.69		
Degrees of freedom	27		
p-value	0.00		

Table 3. Birth Intervals by Time Since Last Birth, Survival of the Previous Infant, Type of Mother, and Type of Birth, Henry Family Reconstitutions, France, 1670-1829

	Previous infant survived 9+ months*			Previous infant died <9 months*		
	Mother of singletons only	Mother of twin		Mother of singletons only	Mother of twin	
Years since last birth	Singleton	Singleton	Twin	Singleton	Singleton	Twin
.6-1	1.2	1.3	1.6	8.5	10.0	21.2
1-1.25	5.2	6.4	4.7	22.9	23.6	24.0
1.25-1.5	7.8	9.6	5.8	19.3	18.6	11.6
1.5-2	21.3	24.6	18.3	22.9	22.2	20.6
2-3	38.9	38.7	41.7	17.6	16.8	13.7
3-4	14.8	12.3	14.8	5.3	5.7	3.4
4+	10.8	7.1	13.1	3.6	3.1	5.5
Total	100.0	100.0	100.0	100.0	100.0	100.0
Mean	2.61	2.38	2.73	1.80	1.75	1.67

* For twins, we use the date of death of the child with the longest life.

Table 4. Effects of Covariates on the Relative Risk of a Birth for Married Women by Period, Henry Family Reconstitutions, France, 1670-1829

	Model 1		Model 2		
	Relative risk	p-value	Relative risk	p-value	Mother had twin by period
Mother had twins	1.05	0.04	1.06	0.24	
Period					
1675-1699	1.02	0.74	1.02	0.78	
1700-1724	1.04	0.17	1.02	0.46	
1725-1749	1.03	0.16	1.02	0.26	
1750-1774	1.00	ref.	1.00	ref.	
1775-1799	0.95	0.01	0.96	0.04	
1800-1829	0.71	0.00	0.71	0.00	
Mother had twin x Period					
1675-1699			1.02	0.91	1.08
1700-1724			1.22	0.03	1.29
1725-1749			1.04	0.56	1.10
1750-1774			1.00	ref.	1.06
1775-1799			0.90	0.15	0.96
1800-1829			0.95	0.47	1.00
Age					
15-19	0.96	0.45	0.96	0.44	
20-24	1.05	0.03	1.05	0.03	
25-29	1.00	ref.	1.00	ref.	
30-34	0.88	0.00	0.88	0.00	
35-39	0.60	0.00	0.60	0.00	
40-44	0.21	0.00	0.21	0.00	
45-49	0.00	0.00	0.03	0.00	
Married less than 5 years	1.29	0.00	1.29	0.00	
Previous infant alive	0.50	0.00	0.50	0.00	
Region					
Northeast	1.00	ref.	1.00	ref.	
Northwest	0.87	0.00	0.87	0.00	
Southeast	0.85	0.00	0.85	0.00	
Southwest	0.72	0.00	0.72	0.00	
Births	23584		23584		
Time at risk	80681.8		80681.8		
Likelihood ratio chi squared	7903.8		7915.8		
Degrees of freedom	17		22		
p-value	0.00		0.00		

Table 5. Effects of Covariates on the Relative Risk of a Birth for Married Women by Period by Region, Henry Family Reconstitutions, France, 1670-1829

	Northeast			Northwest			Southeast			Southwest		
	Relative risk	p-value	Mother had twin by period	Relative risk	p-value	Mother had twin by period	Relative risk	p-value	Mother had twin by period	Relative risk	p-value	Mother had twin by period
Mother had twins	1.21	0.03		1.14	0.21		1.03	0.74		0.72	0.05	
Period												
1675-1699	0.82	0.14		1.09	0.34		0.99	0.90		1.25	0.30	
1700-1724	0.93	0.11		1.00	1.00		1.13	0.01		0.94	0.60	
1725-1749	1.03	0.47		0.93	0.12		1.08	0.05		1.06	0.40	
1750-1774	1.00	ref		1.00	ref		1.00	ref		1.00	ref	
1775-1799	0.98	0.50		0.93	0.09		0.97	0.32		0.96	0.43	
1800-1829	0.65	0.00		0.67	0.00		0.82	0.00		0.76	0.00	
Mother had twin x Period												
1675-1699	0.88	0.71	1.07	0.84	0.44	0.95	1.93	0.16	1.98			
1700-1724	1.16	0.29	1.41	0.91	0.58	1.03	1.52	0.02	1.56			
1725-1749	1.03	0.80	1.25	0.84	0.22	0.95	1.01	0.93	1.04	1.70	0.04	1.22
1750-1774	1.00	ref	1.21	1.00	ref	1.14	1.00	ref	1.03	1.00	ref	0.72
1775-1799	0.72	0.02	0.87	0.84	0.23	0.95	1.07	0.55	1.10	1.09	0.70	0.78
1800-1829	0.83	0.10	1.00	0.95	0.76	1.08	1.10	0.46	1.13	1.25	0.40	0.89
Age												
15-19	1.04	0.75		1.05	0.70		1.00	0.98		0.89	0.28	
20-24	1.03	0.47		1.08	0.10		1.07	0.06		1.11	0.06	
25-29	1.00	ref		1.00	ref		1.00	ref		1.00	ref	
30-34	0.87	0.00		0.88	0.00		0.87	0.00		0.93	0.14	
35-39	0.59	0.00		0.59	0.00		0.60	0.00		0.62	0.00	
40-44	0.20	0.00		0.21	0.00		0.21	0.00		0.22	0.00	
45-49	0.02	0.00		0.02	0.00		0.07	0.00		0.01	0.00	
Married less than 5 years	1.25	0.00		1.36	0.00		1.26	0.00		1.29	0.00	
Previous infant alive	0.51	0.00		0.45	0.00		0.51	0.00		0.47	0.00	
Births	8039			4911			7898			2736		
Time at risk	25701.3			16693.4			27265.1			11022.0		

Likelihood ratio chi squared	2873.2	1795.9	2280.2	785.4
Degrees of freedom	19	19	19	17
p-value	0.00	0.00	0.00	0.00

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