

Chapter 11

The Impact of Intergenerationally-Transmitted Fertility and Nuptuality on Population Dynamics in Contemporary Populations

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Overview: There are intergenerational continuities in contemporary fertility, mortality and partnership behaviors due to genetic and environmental factors. If persistent, these would be expected over time to lead to a proportionate increase in those with a higher than average propensity to reproduce, and consequently to lead to higher population growth (or lower decline) than would otherwise be the case. We use three scenarios of fertility transmission to investigate the differences in long run population dynamics under models of intergenerationally correlated fertility and partnership behaviors:

- (1) fertility is not heritable;
- (2) daughters' fertility is partly correlated with mother's fertility;
- (3) daughters have the same fertility propensity (fecundability) as their mothers.

Positive correlations increase population growth rates substantially, even though the correlation coefficients between completed fertility of mothers and daughters may be modest with the assumptions of these models. This suggests that large samples are required to detect such effects in historical populations, and that the widely-held assumption that fitness was not heritable is questionable. The demographic regime is based on that of England and Wales from 1750 to 2050 and so covers typical long-term experiences of now-developed societies as they moved from pre-transitional to contemporary patterns of below-replacement level cohort fertility. The methodology is based on microsimulation of full kinship networks based on the Berkeley SOCSIM program.

1. Introduction

A standard result in demographic analysis is that given two initial populations with different rates of growth, no matter how small the advantage, the one with the higher value will come to dominate numerically the lower one and the population eventually becomes effectively homogeneous and consists only of the higher growth population (e.g. Keyfitz 1985, Section 1.4). Of course, there must be some mechanism that transmits the rate of growth between generations, whether genetic, environmental or an interaction of the two, and presumably these mechanisms remain unchanged over time if the rate of growth does so also. A similar result – that any population with an enduring advantage in reproductive success will come to dominate numerically – is found in Mendelian genetics, namely Fisher's fundamental theorem of natural selection (Fisher, 1930) which states that any trait correlated with fitness should have a heritability¹ of zero, essentially for the reason set out above. On the other hand, observational studies show that there can be very large variability in fitness: for example, species such as the sage grouse in which more attractive males have hugely greater reproductive success in mating, producing the so-called 'lek paradox' (Boyce, 1990; Pomiankowski & Moller, 1995). The status of Fisher's fundamental theorem of natural selection has been considerably elucidated in recent years (Price, 1972; Edwards, 1994; Frank 1988), and two main sets of explanations for persistent variability in fitness have been advanced, those to do with a host-parasite 'arms race' (Hamilton & Zuk, 1982) and the role of genetic mutation (Kondrashov, 1988; Houle, 1998).

Fertility is a major component of fitness and it is therefore plausible that it should not be an inherited trait in the sense above, at least under a number of restrictive conditions. Empirical analysis of (human) historical populations has shown that the correlation of fertility between generations is close to zero. However, studies of more recent populations have indicated that there is now a correlation coefficient value of about 0.2 averaged across a number of different studies (Murphy, 1999). More specific studies based on kinship behavior genetic models and twin studies have found a relatively strong heritable component of fertility and related behaviors, including nuptiality and marital breakdown in more recent periods (McGue and Lykken, 1992; Dunne et al, 1997; Kohler, Rodgers, and Christensen, 1999; Rodgers and Doughty, 2000; Kirk et al, 2001): for a good summary of recent work, especially in relation to Fisher's fundamental theorem, see Rodgers, Hughes et al (2001).

There are a number of reasons why the superficially incompatible results for historical and recent/contemporary populations differ². These include

1. Fisher's fundamental theorem assumes essentially long-term stability by a large population in an unchanged environment with non-assortative mating and Mendelian inheritance.
2. Fitness rather than fertility is the key indicator - in demographic terms, net reproduction rate (NRR) not gross reproduction rate (GRR).
3. Historical studies may not be of sufficiently high quality or of sufficiently large size to detect intergenerational effects because the techniques used such as family reconstitution are rarely able to follow populations over long periods of time, or consist of genealogies of atypical populations such as aristocratic lineages.

For point 1, such conditions may be too restrictive to be observed in practice³. In addition, it would also be possible for a genetic predisposition to remain uncorrelated with fitness in earlier generations, but to emerge in a different environment. In particular, there is now greater scope for individual choice in fertility outcomes: in the past, sex and reproduction were closely linked, so those with a strong preference for sex, but who were relatively indifferent to children might be expected to have similar family sizes to those with these preferences reversed, but this will not necessarily be the case in contemporary societies when sex has become largely detached from reproduction, so that the former without the latter is readily available (Rodgers, Kohler et al, 2001).

For point 2, if those from large families were less likely to survive or to reproduce if they did survive, then they might have no reproductive advantage. Such a statement is uncontentious in non-human studies where much attention has been given to the inverted-U shape of fitness and the optimum Lack clutch size. For humans, this may be illustrated by the fact that although (dizygotic) twinning has an inherited component, albeit weak, the higher mortality of twin offspring has in the past offset their higher fertility in harsh environmental conditions (Lummaa et al, 1998). However, as the survival difference between single and multiple births decreases (which could happen even if the mortality ratio of twins to singletons increases), *ceteris paribus* this would be expected to lead to an increasing proportion of multiple births in future. A similar finding holds for other partially-inherited variables such as birth weight, birth order and maternal age which were formerly associated with substantial infant mortality differentials, but these differences in mortality and survival have now been considerably reduced (Ulizzi, Astolfi & Zonta, 1998). In contemporary developed societies, mortality rates are so low that parity-specific differentials in survival are trivial and fertility may be treated as equivalent to fitness (Charlsworth, 1980). The idea that we are moving to a higher Lack

clutch size as the dominant model for interpreting fertility change seems highly unconvincing.

Point 3 is an empirical question that will be addressed later.

It is perhaps tempting to assume that 'genetic' effects will be more persistent than 'environmental' ones, and therefore to be more important in discussion of long-term population change, but this is not necessarily the case: under heavy selection pressures, the genetic composition of the population can change substantially over shorter periods (Cavalli-Sforza and Bodmer, 1971). Moreover, the empirically estimated magnitude of the additive genetic effect for fertility has shown considerable short-term movements, for example, from over 50% to under 10% in Denmark for female twin cohorts born around ten years apart around 1890 (Kohler, Rodgers, & Christensen, 1999). However, for the viewpoint of the implications for population dynamics, the relative contributions of nature and nurture to such correlations are largely irrelevant - even if such effects may be distinguishable in specific circumstances, such as by comparing the fertility of monozygotic and dizygotic twins, of kin of different degrees of relatedness, or of adopted and non-adopted children. The key point is that if inherited factors exist that are correlated with reproductive fitness, and if this association continues into the future (as would be expected if the future is sufficiently like the present), this will have implications for population size and structure. This paper aims to quantify the magnitude of such effects on population dynamics in contemporary developed society populations in Section 3. Before doing this, however, we consider the nature of inherited factors and how they are related to actual fertility in Section 2.

2. Fecundability and Fertility

Murphy and Wang (2001) investigated the magnitude of correlations of fertility between successive generations using a simple model of inheritance. If the distributions of parental and children's family sizes are distributed as Poisson with a mean of 2.5 children, and if the children's Poisson distribution parameter is $2.5 + \lambda * (2.5 - \text{parent's no. sibs})$, then the expected numbers of children and sibs in each generation is about 2.5, and the correlation coefficients between number of sibs and children in successive generations is almost identical to λ for λ less than 0.3 in absolute value; for example, with a value of $\lambda = 0.2$, if the parental number of sibs was 5, the child's expected family size would be distributed as Poisson with a mean of 3 children.

Such a model shows the magnitude of effects required to produce a given intergenerational fertility correlation. An intergenerational correlation of about 0.2 is typical of values found in practice, but for genetic transmission between generations of inherited characteristics, achieved

parental family size is not necessarily the best starting point. Sibship size is, at least in part, a socialization, environmental or life course variable since it affects an individual's childhood circumstances, including through mechanisms such as education or standard of living, and consequently it may affect the child's later behavior including subsequent childbearing. These standard social science mechanisms have often been assumed to be the ways in which intergenerational continuities in behavior arise (Thornton, 1980).⁴ However, the underlying Poisson parameter, the reciprocal of the instantaneous birth rate, rather than achieved fertility, is the potentially heritable component since achieved fertility is partly random (and in the real world, determined by a variety of life course factors also). Indeed, in the model above, in the first generation, every parent has the same underlying Poisson parameter, and therefore, there is no genetic variability (see footnote 1).

The natural starting point for investigating models of intergenerational transmission of fertility behavior is that of heterogeneous probability of giving birth. The monthly probability of conception or birth (depending on the context) to a woman at risk of the event is referred to as fecundability (Gini, 1924; Leridon, 1977). We therefore consider models in which this underlying propensity to give birth may be intergenerationally transmitted.

3. Simulation models under alternative assumptions

We generate a number of long-run simulations using the SOCSIM microsimulation program, in which appropriate monthly probabilities of fertility, mortality and nuptiality are applied to an initial population of size 10,000 (Hammel, Mason, & Wachter, 1990) using empirically derived values for each series from 1750 to 1990. For 1990 to 2050, broadly similar values to the period 1980-1990 have been used. Fertility varies by age, marital status and parity. These baseline values represent our best estimate of how demographic parameters have varied in Britain since 1750, further details are given in Murphy (2001). In order to allow for heterogeneity between women, a random lifetime fertility multiplier is generated distributed with an approximate beta distribution with mean one, variance of 0.416 (coefficient of variation of 0.645), maximum of 2.4 and minimum of zero, as suggested by Wachter (n.d.). An individual woman's monthly birth probability is given by the appropriate baseline distribution multiplied by this random variate. Since the mean of the distribution is one, the fertility rates of the group of women are equal to the baseline input rates.⁵ Such a choice produces distributions of fertility that are close to that empirically observed baseline values, but with greater and more realistic variability in the fertility distribution than is the case for a uniform distribution, and, in

particular, it permits the role of intergenerational transmission of heterogeneity to be assessed. In most analyses, fecundability is also assumed to follow a beta distribution (Barrett, 1971; Wood & Weinstein, 1988). In our analysis, we do not identify periods of susceptibility to pregnancy, apart from a minimum gap of 12 months between births, but we assume a similar distribution of heterogeneity between women. We therefore call this fertility multiplier, which gives the relative probability of a woman in a particular age, marital status and parity category giving birth in a given month, 'naïve standardized fecundability' (NSF) -- naïve because it subsumes all the other proximate determinants (Bongaarts & Potter, 1983) and standardized because the mean value is one. As with the usual definition of fecundability, it obscures some aspects of the reproductive process but the present formulation has some advantages in permitting concentration on intergenerational continuities.

If there is no inherited component to fertility, the mean value of naïve standardized fecundability will remain one, and the population fertility rates will be equal to the baseline values at that time period. However, if there is inheritance, then on average more fecund women will have larger numbers of daughters who will themselves have higher values of NSF and form a higher proportion of their generation, so that the mean value of NSF will increase generation by generation (we assume that such correlations will be positive, as found empirically, but negative correlations are also theoretically possible). The overall level of fertility will be higher than in the absence of such transmission because it is given by the original baseline value multiplied by the average population value of naïve standardized fecundability⁶.

A similar heterogeneity multiplier is used to adjust the baseline nuptiality and marital breakdown rates since there is known to be intergenerational transmission of such behaviors. Intergenerationally-transmitted naïve standardized nuptiality (NSM), will further tend to inflate the correlation of fertility between mothers and daughters since early-marrying mothers will spend more of their fertile period in the higher-fertility married state, and thus have more children than average, who will in turn have more children because of their propensity to marry young (although historical data suggest a relatively low intergenerational correlation for age at marriage, Levine, 1982). The effect of increased propensity to suffer marital breakdown might be expected to be less substantial. We assume that the multipliers for fertility, nuptiality and marital breakdown are independent.

In this application, the model of fertility inheritance is that the value of naïve standardized fecundability of a daughter, f_d is given by

$$\begin{aligned} f_d &= f_m && \text{if } Pr(\mathbf{R} \leq \alpha) \\ &= f && \text{if } Pr(\mathbf{R} > \alpha) \end{aligned}$$

where \mathbf{R} is a uniform random variate in $[0,1]$, f_m is the mother's NSF value and f is an independently drawn value from the beta distribution as before. Alpha can run from zero (no intergenerational transmission) to one (each daughter has exactly the same naïve standardized fecundability as her mother). If there is inheritance, then the mean of the naïve fecundability distribution will increase but the variance will remain largely constant (it will also be affected to some extent by the shift in the mean).⁷

In order to show the effect of inheritance, we show results for three assumptions:

1. naïve standardized fecundability is independent between generations (i.e. alpha is set to 0);
2. partial inheritance, with alpha set to 0.5;
3. full inheritance, with alpha set to 1.

We include non-zero values of alpha from 1880 only since the values of the inherited fertility and nuptiality in pre-transitional periods appear to be close to zero, a point we return to later.

Figure 1 shows the effect of these alternative inherited values. The same baseline demographic parameters are used in all runs, and they differ only in that there are different levels of fertility and nuptiality transmission through generations. In order to concentrate on the effect of these changes, we use the same random seeds for the three runs so the values in the first part of the simulation are equal. We use the same model of transmission for nuptiality and marital breakdown as for fertility, with the same values of alpha. Thus mothers with a high (or low) propensity to form or dissolve partnerships will pass on this trait moderately or strongly to their daughters in cases 2 and 3 respectively.

The effect of wholly transmitted regime is substantial. The TFR is well over twice as high by 2050 compared with the non-inherited model (Figure 1(a)). The partially inherited model gives a value that is much closer to the independence model, but still larger by about 300 per 1,000 women by 2050, and it produces a series of TFR values that are around replacement level in 1990, whereas the independence values are well below it in the later part of the period. The annual number of births is also much larger in the wholly inherited case, so that the population size after 170 years of wholly inherited fertility is about fourteen times as large with full transmission as without it. The partial inheritance model gives a value that is much closer to the independence model, but still about 2.5 times larger (Figure 1(b)).

If nuptiality is inherited, in successive generations those who marry early will have more children on average and produce a greater proportion of the population in the next generation, since marital fertility is higher than non-marital fertility and hence the average nuptiality propensity will increase even in the absence of inherited fertility.⁸ The effect of wholly

Figure 1a: Total Fertility Rate by Level of Inheritance Model

Figure 1b: Population size by Level of Inheritance Model

Figure 1c: Female Average Age at Marriage by Level of Inheritance Model

Figure 1d: $e(0)$ for Females by Level of Inheritance Model

inherited nuptiality is to reduce average age at first marriage by about 3.4 years by the end of the period (Figure 1(c)), and the effect of partial transmission is to reduce it by 1.4 years. Variables that are largely uncorrelated with fertility should not show any difference in these cases, and this is confirmed for mortality experience, which does not differ between these models, and so mortality differentials cannot contribute to the observed differences in fitness or population size (Figure 1(d) – we do not consider mortality-fertility correlations in this paper). The same lack of association with fitness also holds for an inherited propensity to experience marital breakdown, because fertility differentials between high and low breakdown propensity groups is small, even though genetic and other inherited components have been established (Pope and Mueller, 1976; McLanahan and Bumpass, 1988).

Table 1 shows how the values of summary demographic indices including the means and standard deviations of naïve standardized fecundability and nuptiality change under alternative inheritance regimes. With a wholly inherited regime, the average level of naïve standardized fecundability is about 2.0, and naïve standardized nuptiality is about 1.7 by the final period 2000-2050, after about 170 years of inheritance. The impact of lower levels of inheritance is less, but there is still a tendency for fecundability, and to a lesser extent nuptiality, to increase. The standard deviation of the independence and part inheritance models remain largely unchanged, but the variances in the full inheritance model decline even though the mothers and daughters have the same values, in the case of NSF by 75% by 2000-50.

Overall fertility measures such as the TFR are sensitive to values of both nuptiality and marital-specific fertility rates. In order to decompose the effects of these two components, we also show in columns 5 and 6 of Table 1, the mean values with inherited nuptiality with alpha equal to 0.5 and non-inherited marital-specific fertility rates and *vice versa*. Columns 2 and 3 give the values with no and both inherited components with alpha equal to 0.5: the average level of fertility (TFR) is 40% higher in the latter case in 2000-50, with about one quarter due to inherited nuptiality, and three quarters due to inherited fertility.

The models above are not meant to be realistic, especially the full inheritance model which produces values that are very different from any contemporary developed society population and has an extreme behavioral assumption: rather they are designed to span the range of possibilities and to quantify the magnitude and implications of different inheritance mechanisms. In order to make comparisons with empirical results, we computed the most widely used index of intergenerational fertility continuity, the Pearson product moment correlation coefficient of completed fertility of mothers and daughters, for which there are a large number of

Table 1: Mean values of naïve standardised fecundability (NSF), naïve standardised nuptiality (NSM), and total fertility rate (TFR) under alternative inheritance regimes

Variable	Independent (alpha = 0)	Partial inheritance (alpha = 0.5)	Full inheritance (alpha = 1.0)	Fertility only inherited (alpha= 0.5)	Nuptiality only inherited (alpha= 0.5)
NSF					
1850-99	1.01 (0.64)	1.09 (0.65)	1.17 (0.66)	1.09	1.01
1900-49	1.00 (0.64)	1.26 (0.66)	1.68 (0.53)	1.28	1.00
1950-99	0.99 (0.64)	1.28 (0.68)	1.92 (0.40)	1.30	0.99
2000-49	0.98 (0.64)	1.32 (0.69)	2.03 (0.32)	1.33	0.99
NSM					
1850-99	1.00 (0.65)	1.05 (0.65)	1.11 (0.65)	1.00	1.06
1900-49	1.00 (0.65)	1.16 (0.65)	1.42 (0.59)	1.00	1.13
1950-99	1.00 (0.64)	1.16 (0.65)	1.58 (0.55)	1.00	1.14
2000-49	0.99 (0.65)	1.19 (0.66)	1.74 (0.50)	1.00	1.18
TFR (per 1,000)					
1850-99	4,003	3,971	3,995	3,998	4,023
1900-49	2,174	2,589	3,350	2,549	2,290
1950-99	1,770	2,265	3,333	2,213	1,877
2000-49	1,315	1,862	3,237	1,740	1,541

Note: standard deviations for selected values are given in brackets.

published values for comparison (Murphy 1999). We confine our calculated values to cases in which both mothers and daughters were alive at age 45 to remove the effect of censoring of childbearing due to death (Table 2): in order to improve precision, we use an initial population of size 40,000. With the intermediate level of transmission of alpha equal to 0.5 for NSF and NSN, the correlation in completed fertility is only about 0.13 for daughters born in the twentieth century (the age 45 requirement by the end date of 2050 means that information is not available for daughters born beyond 2005). What is striking about these fertility correlations is that they are considerably lower than values found in actual contemporary populations, which are typically of the order of 0.2 (Murphy, 1999). These model correlations are low, but this is because the correlation between fertility of mothers and daughters arises only through the correlation of fecundability of mothers and daughters. If the correlation between fertility and fecundability of a woman is β , and the correlation between fecundability of mothers and daughters is δ , then the correlation between fertility of mothers and daughters is $\delta\beta^2$ (as shown in the Appendix). The correlation between NSF and fertility for mothers is usually rather higher than for daughters because mothers are a selected group who have given birth to at least one daughter it is also larger in the earlier period when fertility is higher. Empirically, values of β are around 0.5 with the models used here (Table 2) and are essentially independent of δ , so that even with the implausible value of 100% transmission of fertility, ie $\delta=1$, the correlation between mothers and daughters would only be β^2 and hence about 0.25, or similar to values found in practice.

A major assumption of the analysis here is that the coefficient of variation of the NSF and NSM distributions is about 0.6, since this value determines β . Since the intergenerational fertility correlation coefficient is proportional to β^2 , it is therefore highly sensitive to the assumed variability of NSF (and to a lesser extent NSN also). While good estimates of biological fecundability exist (Wood and Weinstein, 1988), estimation of variability is more problematic, since, for example, a declining hazard rate of conception may arise from population heterogeneity or from duration dependence (possibly due to factors like declining coital frequency rates).

The implication of this analysis may be illustrated by considering the results of Langford and Wilson (1985, p 442) who concluded that 'it is highly implausible that there truly is an association between daughters and mothers in fecundity' using English parish record data. While this conclusion was based in part on additional analyses such as the patterns within individual parishes, a principal reason was because the estimated correlation coefficient of 0.021 of children born to 10,931 mother-daughter pairs was not statistically significantly different from zero, since the 95%

Table 2: Correlation coefficients of naïve standardised fecundability (NSF), naïve standardised nuptiality (NSM), and completed family size (parity) under an intermediate inheritance regime ($\alpha = 0.5$) for women surviving to at least age 45.

Period of birth*	Variable	Individual correlations [†]			Mother-daughter correlations [‡]		
		NSF	NSN	Parity	Mother's NSF	Mother's NSN	Mother's Parity
1850-99							
(N=27,802)	NSF	-	0.01	0.52	0.21	0.00	0.12
	NSN	-0.01	-	0.32	0.00	0.22	0.02
	Parity	0.70	0.16	-	0.09	0.04	0.08
1900-49							
(N=39,861)	NSF	-	-0.01	0.57	0.42	0.00	0.21
	NSN	-0.01	-	0.24	-0.02	0.47	0.05
	Parity	0.50	0.11	-	0.23	0.10	0.12
1950-99							
(N=52,894)	NSF	-	-0.01	0.49	0.42	-0.01	0.23
	NSN	-0.02	-	0.29	-0.01	0.48	0.07
	Parity	0.53	0.13	-	0.19	0.12	0.13

Notes:

* birth cohort is that of the women in individual-level correlations, and that of the daughter for mother-daughter pair correlations. The sample size refers to all women born in period surviving to age 45.

[†] all women's (ie daughters') values shown above diagonal: mothers' values shown below diagonal.

[‡] columns refer to daughter's and rows to mother's values, both of whom survive to age 45.

confidence included the value of zero (there was clear evidence of incompleteness in these data and a coefficient of 0.033 was found for a sub-sample of 939 women for whom there was more complete information, but even here the data contained some errors, and this is therefore probably an underestimate to some extent). For the reason set out above, the maximum correlation in a natural fertility population is β^2 , which would be about 0.36 with the assumed coefficient of variation for NSF (the correlation between NSF and fertility is higher in pre-transitional populations, at about 0.6), but would be less if the coefficient of variation of NSF was smaller: with the present distribution, the fertility rate for a woman at the first quartile of the distribution would be just under one third of one at the third quartile. For

historical populations, the estimated value of beta, 0.6, is coincidentally the same as the coefficient of variation of the NSF distribution used here, and with a coefficient of variation of 0.3 (detailed values not given here), the value of beta was found to be about 0.3, and, of course, it is zero when beta is zero. Thus the maximum intergenerational fertility correlation is approximately equal to the coefficient of variation of the NSF distribution, at least for values below 0.6. Consequently, the *estimated intergenerational fertility correlation coefficient* implies an estimate of *inherited fecundability of mothers and daughters* in this historical population which is much higher than the estimated value of 0.033 (which is probably an underestimate as noted above) with the assumptions of the model here, albeit with a high standard error, but the actual magnitude depends on the unmeasured coefficient of variation of the NSF distribution: if it was 0.3, then the intergenerational fecundability correlation coefficient would be 0.37, and if it was 0.6, the value would be 0.09. We would therefore question the widely-held assumption that the low intergenerational fertility correlations found in historical data show non-heritability of fitness and so provide empirical support that Fisher's fundamental theorem, and that the topic requires further investigation.

Although we have based our analyses on the assumption that fecundability was not inherited before 1880, the date usually taken as marking the fertility transition in England and Wales as well as in many other European societies, we would also note that our results, unlike earlier studies, do not rule out the existence of such effects in earlier periods, although, for the reasons set out above, finding empirical data will be problematic. We also believe that evidence from a range of empirical studies from non-human populations (which have fewer confounding factors and so Fisher's fundamental theorem might be expected to apply more directly) suggest that an inherited component to fertility/fitness variation cannot be ruled out.

Although we have emphasised that our analyses do not distinguish between 'genetic' and 'environmental' transmission mechanisms, nevertheless, we may speculate about the relationship of our findings to the issue of demographic transition, which has been identified as a major problem for evolutionary theories, since populations move from a high fertility to low fertility regime in a relatively short period, when they would often have been able to have had more children due to improved conditions, and bear children at a level below which could have maximised their numbers of children who survive to adulthood. At times when the effective range of options increases, the proportion of variance in fertility due to genetic factors has been found to increase (Kohler, Rodgers, and Christensen, 1999). We believe that the possibility of such factors playing a role in historical population dynamics should now be acknowledged.

However, in earlier periods, the interaction of mortality and fertility cannot be ignored, and further work will be required to elucidate the mechanisms at work.

4. Conclusions and Summary

There are three main points that arise from these findings. The first is that the effect of persistent intergenerational transmission of fertility on population dynamics in contemporary societies is substantial, and possibly among the most important ones that exist. Even with the relatively low levels of transmission of the intermediate case, the population was 2.5 times as large in 2050 as in the case of intergenerational independence. This suggests that more attention should be given to the analysis and interpretation of such trends, because they not only produce large macro-population effects, but also corresponding micro-demographic ones such as the fact that distributions of kin are very unevenly distributed when such patterns of inheritance exist (Murphy and Knudsen, 2002). Inherited fertility not only has a substantial effect on population dynamics in contemporary developed societies, with the sorts of models used here, but since the level of inheritance appears to have increased over recent decades, this role must also be increasing.

The second is that the mechanism used here, namely transmission of naïve standardized fecundability and nuptiality, i.e. a proportionate shift in risk at all ages, is probably not the most appropriate mechanism. While there is evidence that physiological characteristics associated with childbearing, such as age at menarche, have a genetic component (Garn, 1980), such differences may be largely irrelevant when the average age at first birth in some European societies is now close to 30 (Council of Europe, 2001). What is transmitted is not a generalised propensity of potential childbearing (i.e., fecundability), but parity-specific behavior such as a stopping rule or a socialisation mechanism related to reproductive performance (i.e., fertility) rather than fecundability. The transmission mechanism here is based on what may be regarded as physiological differences: further work is needed to elucidate the role of psychological factors.

The third point is that in pre-transitional populations, apart from propensity to marry (which is of less relevance since most studies are based on married mother-daughter pairs), the only plausible mechanism for intergenerational transmission is biological fecundability. The demographically defining characteristic of such populations is that they exhibit natural fertility (Henry, 1961; Bongaarts and Potter, 1983), ie their fertility behavior is independent of number of children already born, although the overall level can vary between populations due to differences

in, for example, breastfeeding practices. Since the childbearing history includes random variability, the question of what is the range of variability that can be plausibly expected becomes relevant. Even with a limiting alpha value of 100%, transmission, correlation coefficients of the order of around 0.10 to 0.35 will be found for the fertility of successive generations, and smaller values of alpha will lead to directly proportional smaller values. This means that very large samples will be required to establish statistical significance, and that alternative research designs such as twin studies may provide additional insights.

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Appendix:

Derivation of Formula for Correlation of Fertility of Mothers and Daughters when Fecundability Is Inherited

u standardised achieved fertility of mother
 v standardised achieved fertility of daughter
 f standardised fecundability of mother
 g standardised fecundability of daughter
 (standardised variables have mean zero and variance of one)

Assume that fecundability and fertility are related as follows

$$u = \beta f + \epsilon_m$$

$$v = \gamma g + \epsilon_d$$

And the fecundability of mothers and daughters is related as follows

$$g = \delta f + \epsilon_i$$

Then

$$\delta u = \delta \beta f + \delta \epsilon_m$$

$$\beta g = \delta \beta f + \beta \epsilon_i$$

$$\delta \gamma u - \beta \gamma g = \delta \gamma \epsilon_m - \beta \gamma \epsilon_i$$

$$\beta \gamma g = \beta v - \beta \epsilon_d$$

$$\delta \gamma u = \delta \gamma \epsilon_m + \beta v - \beta \gamma \epsilon_i - \beta \epsilon_d$$

$$\delta \gamma u - \beta v = \delta \gamma \epsilon_m - \beta \gamma \epsilon_i - \beta \epsilon_d$$

Taking the variance of each side and noting that ϵ_m , ϵ_d and ϵ_i are independent, and that $\sigma^2(u) = \sigma^2(v) = \sigma^2(g) = 1$, and therefore

$$1 = \beta^2 + \sigma^2(\epsilon_m)$$

$$1 = \gamma^2 + \sigma^2(\epsilon_d)$$

$$1 = \delta^2 + \sigma^2(\epsilon_i)$$

then

$$\delta^2 \gamma^2 - 2 \delta \beta \gamma r(u,v) + \beta^2 = \delta^2 \gamma^2 (1 - \beta^2) + \beta^2 \gamma^2 (1 - \delta^2) + \beta^2 (1 - \gamma^2)$$

where $r(u,v)$ is the correlation between achieved fertility of mothers and daughters.

Simplifying gives

$$r(u,v) = \delta \beta \gamma$$

In practice, γ would be expected to be close to β , so further simplifying the formula

$$\text{to } r(u,v) = \delta \beta^2$$

Notes

- ¹ Heritability is usually calculated as a measure of variability in inheritance; for example as the proportion of total variance that is due to inherited factors. Heritability can be zero either because the trait is not inherited, or because there is no genetic variability in the population. Clearly we inherit a propensity to reproduce (Foster, 2000), so the lack of heritability arises from the second reason.
- ² We exclude discussion of the very different results obtained by Pearson and Lee (1899) and Williams and Williams (1974) using apparently the same data on British upper class and noble families. For details, see Murphy (1999).
- ³ We do not pursue the general question of whether a heritable component to fitness would remain in a fixed environment that could arise from host-parasite interactions or from genetic mutation as discussed in the opening section of the paper.
- ⁴ Since a woman's eggs are formed when she was *in utero*, subsequent experience cannot affect her DNA, although it can affect a range of neurological and endocrinological outcomes, especially those associated with her patterns of development as a child, including nurturing which can affect her own childrearing behavior. While childhood experiences as reflected in birth order could theoretically affect the fitness of a gamete, particularly by affecting survival, this variable appears to have little if any effect in contemporary societies (Murphy and Knudsen, 2002). We do not consider effects such as imprinting or epigenetic inheritance that may be responsible for apparently anomalous results such as the particular patterns of inheritance found among survivors of the Dutch Hunger Winter.
- ⁵ This refers to rates within age, marital status and parity. Measures of overall fertility such as total fertility rate (TFR) will also depend on the proportion of time spent in these states. The paper will consider the relative contributions of these two components later.
- ⁶ We do not discuss the contribution of males and females to naïve fecundability in subsequent generations. We have undertaken some experiments with alternative specifications but for simplicity, we concentrate solely – if unrealistically – on female line transmission only.
- ⁷ In the original version of SOCSIM, the basic model of fertility inheritance is that the value of naïve standardized fecundability of a daughter, f_d is given by $f_d = \alpha f_m + (1-\alpha)f$ with the notation as above. Here also alpha can run from zero (no intergenerational transmission) to one (each daughter has exactly the same naïve standardized fecundability as her mother). However, if there is inheritance, the variance will decline. For example, if f_m and f are distributed independently from the same distribution, then if alpha is equal to 0.5 the variance f_d in the next generation will be only about 0.5 of that in the previous one. Therefore the variability of the distribution and hence its influence in subsequent generations will decline.

⁸ In a growing population, earlier childbearing has even greater fitness, but the reverse is true in a below replacement level fertility regime. However, this higher fertility of those who start early is more than sufficient to offset this effect.

Figure 1(a) TFR by Level of Inheritance Model

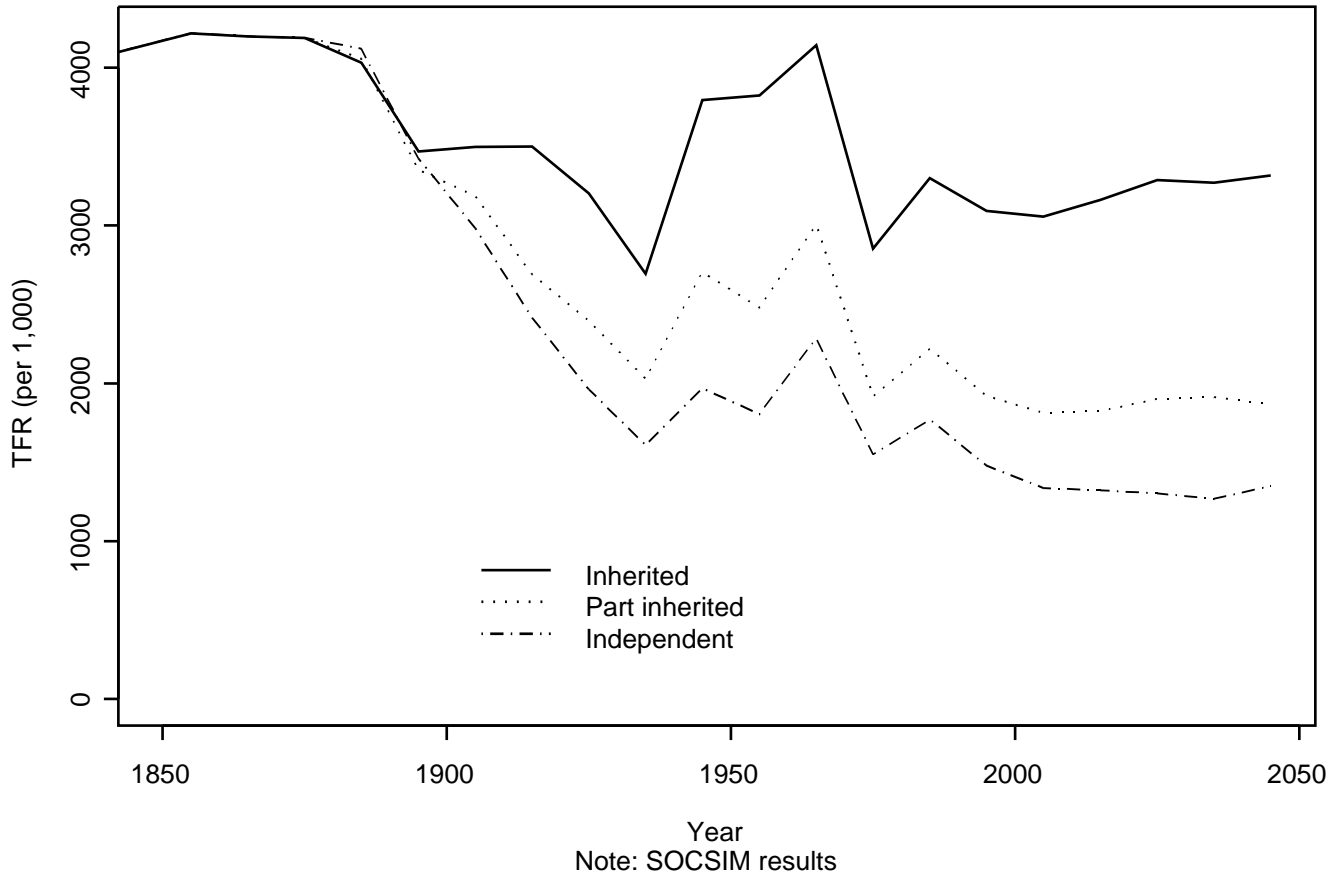


Figure 1(b) Population size by Level of Inheritance Model

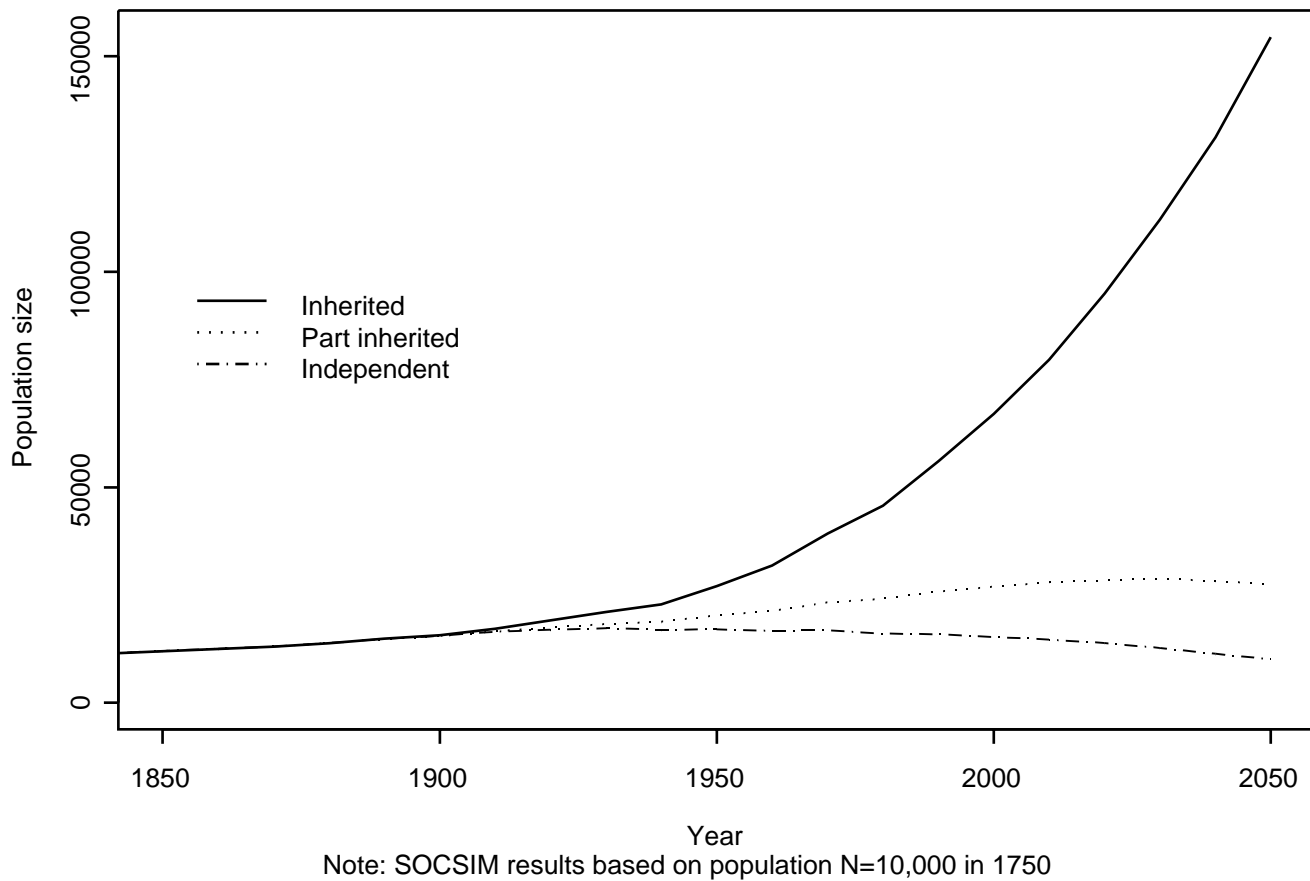
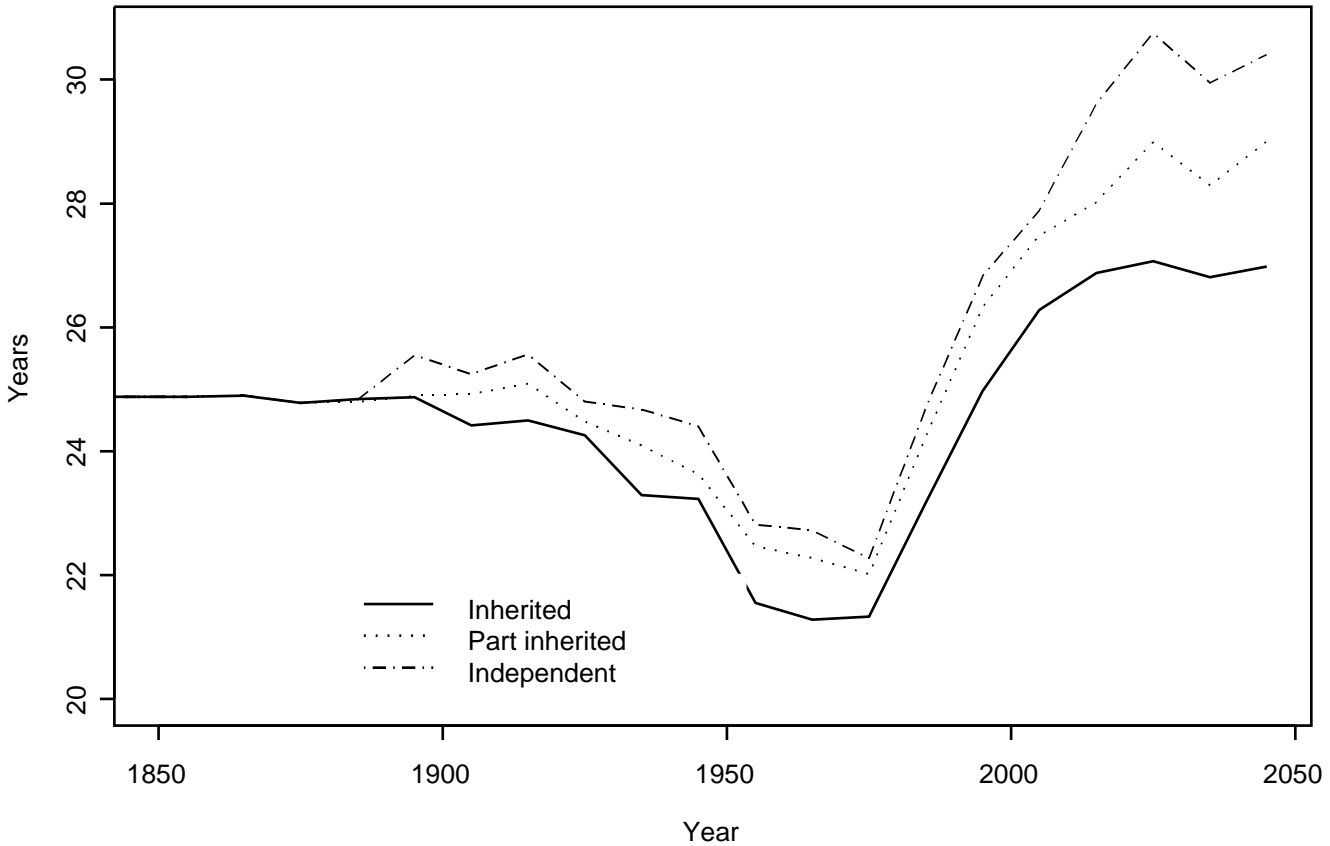
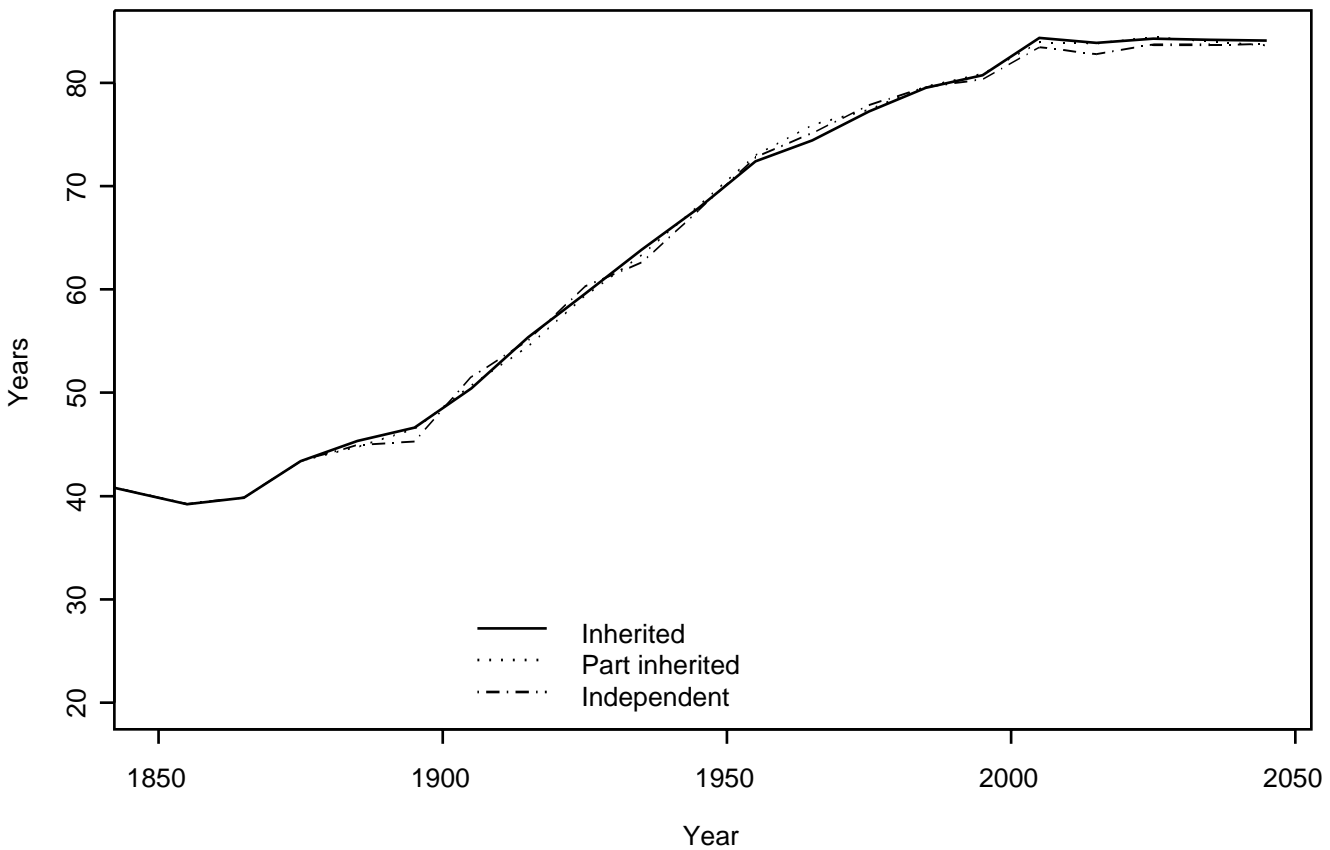


Figure 1(c) Female Average Age at Marriage by Level of Inheritance Model



Note: SOCSIM results based on population N=10,000 in 1750

Figure 1(d) $e(0)$ for females by Level of Inheritance Model



Note: SOCSIM results based on population N=10,000 in 1750