The Changing Nature of the Link Between Infant Mortality and Fertility in Finland 1776—1978

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The issue at task

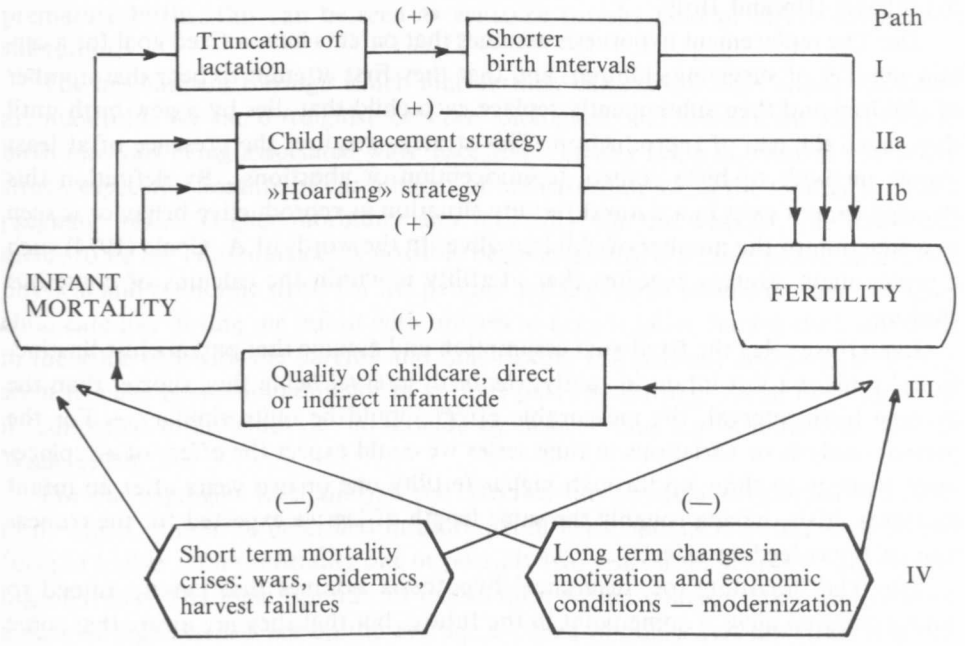
At least since Wappäus published his »Allgemeine Bevölkerungsstatistik» in 1861 the notion that infant mortality is an essential determinant of fertility has become a commonplace in demographic literature. Well before fertility started to decline in Germany, Wappäus had distinguished between a physiological effect of infant mortality via breastfeeding and a voluntaristic one through replacement of dead children. — Almost a century later authors of demographic transition theory (e.g. Heer 1966) saw declining mortality and especially infant mortality as a prerequisite for a fertility decline. Their argument was that parents would have to give birth to fewer children under improved infant and child mortality conditions in order to achieve a certain desired number of surviving offsprings.

While the assumption that fertility is in part determined by the level of infant mortality became widely accepted, and was expressed and analyzed through various hypotheses of child replacement strategies, the possibility that infant mortality might be determined by fertility attracted little attention until recently. At the end of a thorough analysis of links between infant mortality, fertility, and breastfeeding on early German data Knodel and Van de Walle (1967) gave just a short statement on this 'opposite' direction: »One further hypothesis which merits exploration postulates a causal connection in the opposite direction. Perhaps high fertility leads to high infant mortality because parents with large families are less able to give infants the care they require» (Knodel and Van de Walle, 1967, 131). Since then more research was done on this question using family level and regional cross-section data. In the present study, long Finnish time series will be used for an alternative approach to this question; short-term and long-term variations in those data will be exploited for possible information on the nature of the relationship between child mortality and fertility.

Defining possible paths of causation

Before going into the quantitative analysis we will work on an interpretative model and try to distinguish between several possible ways in which infant mortality and fertility can be causally linked. The signs in Figure 1 (+, —) indicate the kind of association that can be expected for each mechanism. This chart does not yet make any assumptions on the timing of the different effects, i.e. on the length of the time lag to be assumed in the quantitative part of this study. The different paths of causation are numbered from I to IV:

Figure 1. Links between infant mortality and fertility



Path I: It seems to be well established that in presence of prolonged lactation the sudden death of an infant and the consequent termination of breastfeeding shortens the period of post partum amenorrhea. Knodel (1978) summarizes the results of studies which had been made for European populations during periods of assumed natural fertility, comparing birth intervals following infant deaths to normal birth intervals. All birth intervals following the death of an infant were significantly shorter except in those areas where breastfeeding was uncommon. This evidence clearly suggests that there was a physiological effect of infant mortality on fertility operating in pre-industrial European populations.

Unfortunately, there is no quantitative information on the extent of breastfeeding in pre-industrial Finland. Pitkänen (1983) suggests that breastfeeding was common in most parts of 18th and 19th century Finland and that the extent of it was subject to regional variation. — For the present model of analysis, however, the lack of detailed information on breastfeeding is not a prohibitive problem, since it only makes it impossible to disentangle the effect due to truncation of breastfeeding and the effect of a possible replacement strategy, as those two effects are parallel (see Figure 1) and roughly simultaneous. For this reason even exact knowledge about the extent of breastfeeding could hardly faciliate a distinction between the biological truncation effect and the behavioral replacement in a macro level analysis of the present kind.

Path II: In most of the recent literature (e.g. Preston 1978, Scrimshaw 1978) possible parental behavior in reaction to infant mortality is seen under two different hypotheses (IIa and IIb):

IIa: The replacement hypothesis assumes that parents have a fixed goal for a certain number of surviving children, and that they first attempt to bear that number of children and then subsequently replace each child that dies by a new birth until they reach the end of reproduction. This strategy requires the presence of at least simple methods of birth control (contraception or abortions). By definition this strategy cannot exist in a natural fertility situation as reproductive behavior is seen as a function of the number of children alive. In the words of A. Coale (1974) such a replacement strategy requires that »fertility is within the calculus of conscious choice».

Even if we relax the fixed goal assumption and assume that parents just 'instinctively' replace a lost infant instantly, the birth interval being thus shorter than the average birth interval, the measurable effect should be quite similar. — For the present analysis of variations in time series we could expect the effect of a replacement strategy to show up through higher fertility one or two years after an infant mortality crisis, having roughly the same length of lag we expected for the truncation of breastfeeding effect.

IIb: The 'hoarding' or 'insurance' hypothesis assumes that parents intend to have x children alive at some point in the future, but that they are aware that some of their children might die before that point but beyond the stage when they are physically able to replace them (see Preston 1978). Therefore parents attempt to have more than x children as an active anticipatory strategy of insurance. This hypothesis does not specify how parents perceive the probability of surviving to a certain age. The perception is probably not based on family experience but rather on a societal level. Furthermore, parental decisions probably do not refer to the child mortality experience at a single point in time or even to a single decade's experience.

Alternative to the strong assumption of individual rationality and personal awareness of child mortality conditions, it could be assumed that there is something like an implicit 'hoarding' strategy, implicit in the social norms about family size and reproduction which have developed over long periods of child mortality ex-

perience. — But, whatever view we take on this hypothesis, it cannot be tested through the analysis of short-term variations of infant mortality and fertility. Its test has to focus on the long-term secular developments of those two variables, a task to be done later in this article.

III: The assumption of a causal path going from fertility to infant mortality is relatively new and still unconventional; the assumption is especially rare in economic analyses of the relationship between fertility and infant mortality. But as mentioned before, there is a number of relatively recent studies showing this kind of effect in historical European situations as well as in contemporary conditions in less developed countries (see e.g. Chowdhury, Khan, Chen 1978). Knodel (1978) using household level data from three Bavarian villages found that the percentage of children dying before age one declines sharply as the length of the previous birth interval increases. Except for the fact that short birth intervals disproportionately consist of premature births, this can be seen as evidence for the kind of effect under consideration.

The mechanisms through which high fertility can lead to high infant mortality are manifold. We can distinguish between 'biological' and 'behavioral' links. Short birth intervals being associated with 'excessive' fertility bring nutritional and other stress, especially because of curtailment of breastfeeding when the mother becomes pregnant again. Higher morbidity and mortality for the weakest, the newborn, seems to be the consequence. A possible behavioral link is related to the additional physical and economic stress for the parents and especially for the mother. Abusive child care like dosing the infant with opiates to keep it quiet, having the baby sleep in the same bed with the parents and thus risking suffocation, and general neglect seem to have been commonplace in various parts of Europe; social historians call it 'concealed infanticide' or 'infanticide by neglect' (see e.g. Knodel and Van de Walle 1979).

For our aggregate level analysis the effect of this possible mechanism of causation, which cannot be described in more detail here, should manifest itself in the form of higher infant mortality one or possibly two years after years of 'excessively' high fertility. The logic behind this expectation is that children born shortly after a year of high fertility are on the average exposed to higher fertility stress (shorter birth intervals) in their families and are thus candidates for nutritional deprivation and 'concealed infanticide'. Conversely, we can expect lower infant mortality for the year following a year of low fertility.

IV: Long term changes in motivation and socio-economic conditions are without doubt important determinants of the secular decline in infant mortality as well as in fertility. For the analysis of relative annual changes, however, these on an absolute scale very important determinants will be omitted from the model because we assume that they do not change from one year to another or change at a slow and steady pace. But, on the other hand, those long term evolutions tend to change the whole structure of the relationship between infant mortality and fertility. For this reason the relationships are estimated separately for different periods corresponding to different stages of modernization.

Short term mortality crises due to wars, famines, and epidemics, on the other hand, were a common feature in premodern societies leaving their marks not only on the time series of mortality but also on those of fertility. Years of extremely high mortality were almost universally also years of extremely low fertility. Already Hotelling and Hotelling (1931) for instance showed for English time series that the variance in births was enormous, much greater than could be expected to arise from random sampling, from economic effects, wars and mobilisation. They concluded that epidemics were the main reasons, leading to profound depressions in births some months later. The mechanisms through which this high negative association worked were probably high foetal mortality due to disease or undernourishment of the mother, induced abortions, and fewer conceptions. Most of these mechanisms work towards a negative correlation between mortality and fertility in the same year (at lag zero).

On methods and data

To eliminate the effect of the secular trends in infant mortality and fertility, which will be discussed later, the input data to this analysis of short-term fluctuations will be annual percentage changes in most models. This transformation of the time series eliminates all linear trends. In our case it eliminates the influence of changing marriage patterns and of long term motivational and socio-economic changes, assuming that they occur at a steady pace during the periods under consideration.[1]

The first method that will be used is that of cross-lagged panel analysis. Since one of the main assumptions of this kind of correlation analysis is stationarity of the causal structure over time (see Kenny 1979), it seems important in this case to calculate the coefficients for different time periods separately. Splitting up a time-series into sub-periods is always somewhat arbitrary. The year 1917 seems to be a reasonable date for a major cutoff point since Finland finished the independence war and became established as a sovereign nation in this year. Around that time many social and economic indicators such as education and level of urbanisation show major changes coinciding with a steep and irreversible fertility decline. For that reason the period before 1917 will be called premodern and that after 1917 modern. As the premodern period seems rather long, a further subdivision was made cutting off in 1860 which is the time when infant mortality started its rapid decline.

[1] The transformation to annual percentage changes of a variable X was done according to the following formula (in our notation a 'PC' is added to the variable's name):

$PCX(t) := (X(t) - X(t-1))/X(t-1)$.

Because the assumption that the different specified lines of causation can be properly estimated by looking at lags zero and one only seems rather strong, in a second step the study will be extended to an analysis of distributed lags.[2]

Another study by Ronald Lee (1981) uses such distributed lag models to analyze short-term variations in vital rates, prices, and weather. His series of births and deaths are monthly data for England 1540—1848. He does not have any information on age-specific rates. In the section analyzing the relationship between fertility and mortality he estimates the net effects of mortality on fertility for the four subsequent years after having eliminated the effect of population size and age structure through removal of long-term variations from the series. He finds that mortality at year t has a significant negative association with fertility at year t and t + 1 and a positive association with fertility at year t + 2. For year t + 3 and year t + 4 the coefficients are slightly negative. Although Lee's method is similar to the one we used, his results do not really help with the question asked here because he has no data on infant mortality and focuses only on the causal path from mortality to fertility.

The annual time series of age-specific fertility rates, infant mortality, and child mortality for 1776—1925 come from Turpeinen (1979) and after 1925 from the Statistical Yearbook of Finland. Since it is not specified by Turpeinen (1979) how the early annual data were derived we assume that the population figures for the denominators of the rates were interpolated over five years because the parish registration forms containing size and structure of the population were only collected every fifth year between 1775 and 1880, whereas the vital statistics sheets were submitted every single year. The coverage of the data is usually regarded to be very good (Turpeinen 1979, Pitkänen 1980).

Before continuing, it has to be mentioned at this point that a potential unknown bias may be present in those models, being caused by the definition of the infant mortality rate which is calculated by taking infant deaths age 0 to 1 in the year t over the number of births in that year. Thus some of the infants dying in year t were born in year t-1 and are therefore not included in the denominator of the infant mortality rate at time t. For this reason strong fertility fluctuations could result in a positive association between infant mortality at year t and fertility at year t-1 which is of purely computational nature and does not reflect any real effect. — But there is reason to assume that this effect is not very strong since the overwhelming part of infant mortality happens within the first half of the first year of life thus reducing the average risk of dying before age one in the calendar year following the year of birth. But still, there remains some uncertainty about this bias, which has to be kept in mind when trying to interpret the results. Further light on this problem can be shed through the analysis of child mortality for age 1—2, because this rate should be free from the bias described above.

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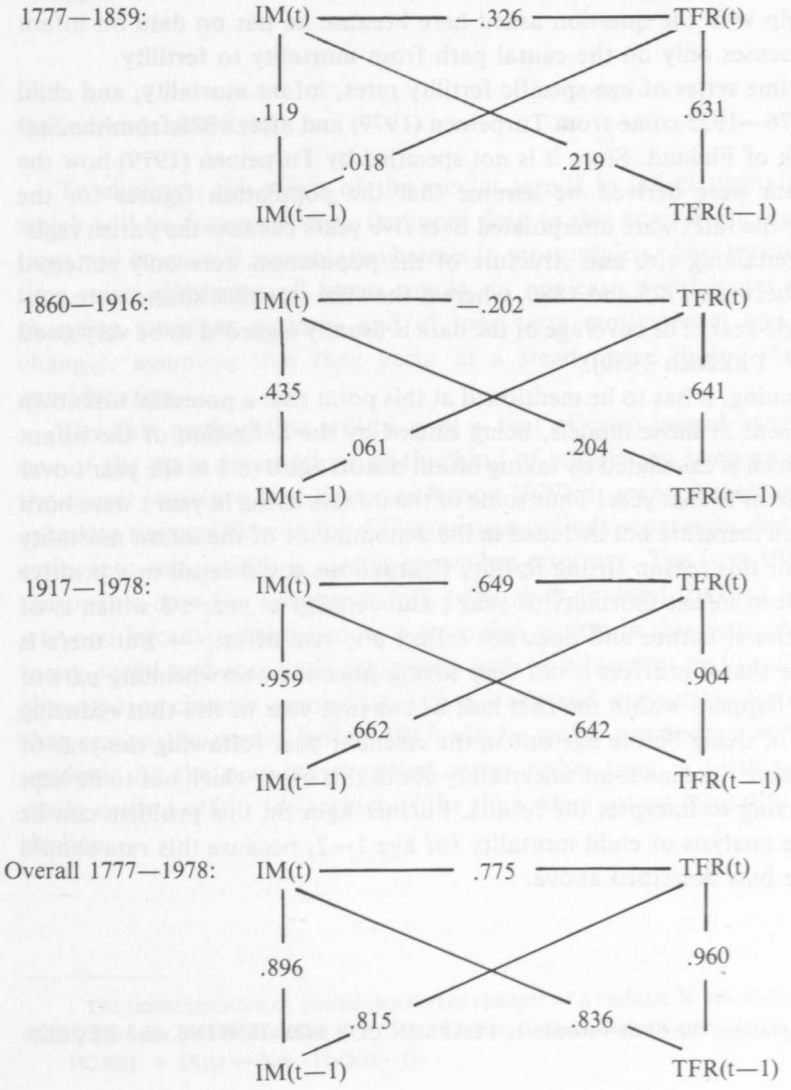
[2] For the computations the SPSS-Procedures PEARSON COP BOX-JENKINS, and REGRESSION were used.

Cross-lagged correlation analysis

Figure 2 gives correlation coefficients between the absolute values of the total fertility rate and the infant mortality rate for simultaneous observations as well as for cases where one of the variables is lagged for one year. To faciliate comparisons the first order autocorrelation coefficients for the infant mortality and fertility series are given, too.

F i g u r e 2. Cross-lagged correlations for absolute values of infant mortality rate (IM) and total fertility rate (TFR).



For 1777—1859 infant mortality and fertility show a significant negative correlation at lag zero. This confirms the expectation that at lag zero exogenous mortality crises cause a negative association between the two indicators. Concerning first order autocorrelations it is worth noting that the coefficient for infant mortality is extremely low, indicating that the annual fluctuations are very strong and almost offset the effect of the already slow decline in that period. The cross-lagged coefficients seem to indicate that there is almost no association between the total fertility rate and the infant mortality rate of the previous year. On the other hand, infant mortality shows a clear positive correlation with total fertility of the year before.

For the period 1860—1916 the coefficients are essentially similar to those in 1777—1859, only somewhat less pronounced. Noteworthy, the first order autocorrelation coefficient has grown to .435 for infant mortality, indicating that the trend has become dominant.

For 1917—1978 the picture is very different, indeed: the correlation between infant mortality and fertility at lag zero became highly positive, thus revealing the decreased importance of acute mortality crises. High first order autocorrelations indicate little annual fluctuations and a strong trend. The cross-lagged correlation coefficients are both highly positive, where the association between current fertility and lagged infant mortality is even the stronger one, thus showing a reversal of the structure prevalent in 1777—1916.

One must be cautious about infering anything from those correlations which are not adjusted for the trend and long-term changes such as those in marriage behavior. For this reason, next, we will do the same kind of cross-lagged correlation analysis for the annual percentage changes in total fertility and infant mortality. The results are given in Figure 3.

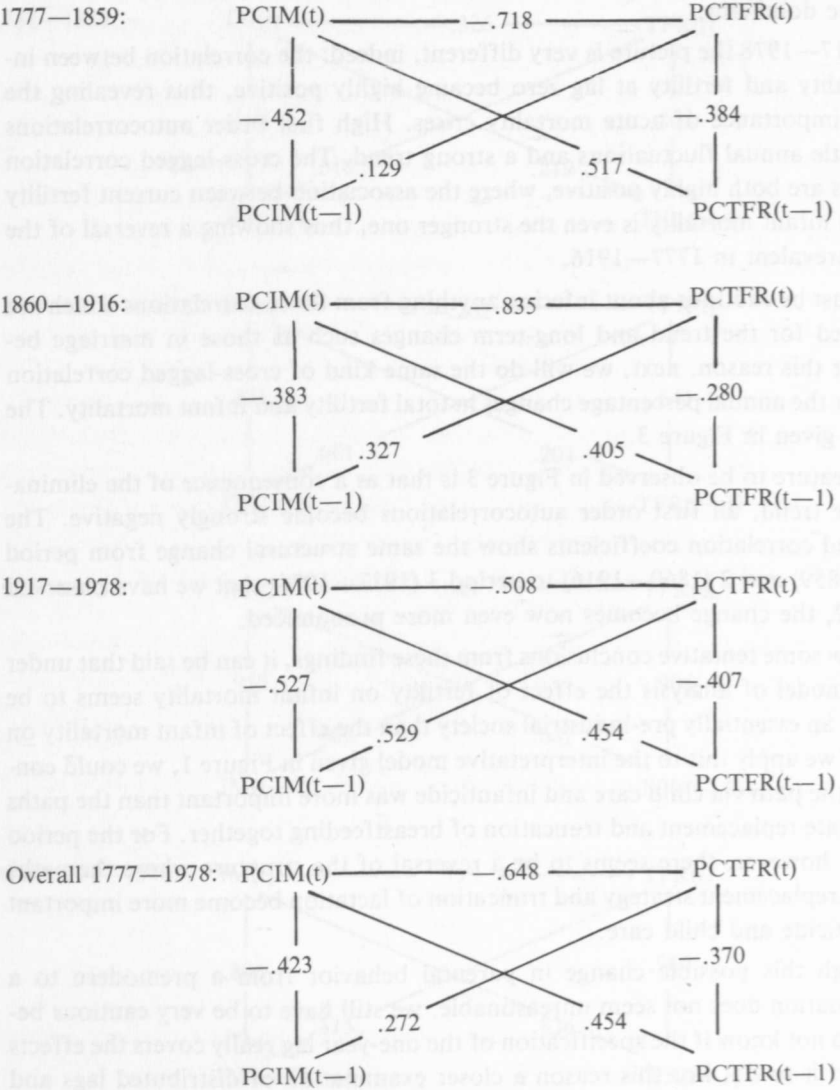
The first feature to be observed in Figure 3 is that as a consequence of the elimination of the trend, all first order autocorrelations become strongly negative. The cross-lagged correlation coefficients show the same structural change from period 1 (1777—1859) and 2 (1860—1916) to period 3 (1917—1978) that we have observed in Figure 2, the change becomes now even more pronounced.

To draw some tentative conclusions from these findings, it can be said that under the given model of analysis the effect of fertility on infant mortality seems to be stronger in an essentially pre-industrial society than the effect of infant mortality on fertility. If we apply this to the interpretative model given in Figure 1, we could conclude that the path via child care and infanticide was more important than the paths via immediate replacement and truncation of breastfeeding together. For the period after 1917, however, there seems to be a reversal of the structure where the paths of rational replacement strategy and truncation of lactation become more important than infanticide and child care.

Although this possible change in parental behavior from a premodern to a modern situation does not seem unreasonable, we still have to be very cautious because we do not know if the specification of the one-year lag really covers the effects we assumed it to do. For this reason a closer examination of distributed lags and

a focus on not only the total fertility rate but also age-specific fertility rates could give us helpful information.

Figure 3. Cross-lagged correlations for annual percentage changes in infant mortality (PCIM) and total fertility (PCTFR)



Distributed lag models

For the following regression models the annual variation in one of the two variables is assumed to be explained by the annual changes in the other variable zero to seven years before. A lag of seven years was chosen as cutoff point after some experimenting with longer and shorter lags. Those trial runs showed that for lags longer than seven years the coefficients became very small and the signs unpredictable; in fact, for most cases this happened already after lag five. We also decided to include the annual percentage changes of the crude death rate at lag zero as an additional independent variable into the equation to control for extreme annual fluctuations due to mortality crises. In the absence of controls for the annual death rate, the almost simultaneous effect of those crises on infant mortality and fertility would lead to unreasonably high coefficients for the explaining variable at lag zero. In the case of distributed lags of fertility change explaining infant mortality variation, for instance, not controlling for the death rate would mean that most of the increase in infant mortality in a year of disease or cathastrophe would be attributed to decreasing fertility, which is not at all reasonable.

Now, of course, the question arises, why not to control for other influences like income, weather, etc. We tried in fact to control for annual changes in income level (from 1870 on when annual data on GDP are available) but there was almost no additional variation attributed to that factor. After all, the annual percentage changes in the crude death rate, PCDR(t), seem to be a reasonably good indicator for annual changes in environmental hazards and economic adversities which cannot be distinguished at this point.

MODEL 1 thus has the annual percentage change in infant mortality at time t, PCIM(t), as dependent variable and the annual percentage changes in total fertility PCTFR(t) ... PCTFR(t—7) plus the death rate change, PCDR(t), as independent variables. In MODEL 2 the explanatory setup is reversed:

$$\text{MODEL 1: } PCIM(t) = A + b1\ PCTFR(t) + b2\ PCTFR(t-1) + \ldots + b8\ PCTFR(t-7) + b9\ PCDR(t) + U;$$

$$\text{MODEL 2: } PCTFR(t) = A + b1\ PCIM(t) + b2\ PCIM(t-1) + \ldots + b8\ PCIM(t-7) + b9\ PCDR(t) + U,$$

where A is the constant and U the error term, assumed to be normally distributed with mean of zero.

The coefficients b1 to b9 can be interpreted as factors by which an increase in the percentage change of total fertility causes an increase in the percentage change of infant mortality, and vice versa. Thus the coefficients are comparable across different models and different periods and their meaning comes very close to that of an elasticity (which is usually estimated by a multiplicative model of the absolute variable values).

Table 1 shows that PCTFR(t) has a significantly negative coefficient even after having controlled for PCDR(t). This negative coefficient at lag zero will show up in all regression models estimated here and is probably due to high incidence of

foetal mortality simultaneous with high infant mortality, famine amenorrhea, or other causes described in the interpretative model. Still in Table 1, the fertility change lagged by one year seems to have a clear positive relation to infant mortality which is especially strong in 1783—1916. This result corresponds with the findings from the cross-lagged correlation analysis.

Table 1. Distributed lag models for annual percentage changes in infant mortality and total fertility for 3 periods.

Regression MODEL 1 (PCIM(t) as dependent variable)

Independent variables:	1783—1916	1917—1978	overall
PCTFR(t—0)	—.401 ***	—.523 ***	—.472 ***
—1	.668 ***	.401 ***	.592 ***
—2	.041	.156	.134
—3	—.082	.067	—.044
—4	—.158	—.098	—.051
—5	—.161	—.363 **	—.157
—6	—.054	.027	.030
—7	—.084	.167	.075
PCDR(t)	.654 ***	.311 ***	.584 ***
	R^2 = .851	R^2 = .540	R^2 = .774

Regression MODEL 2 (PCTFR(t) as dependent variable)

Independent variables:	1783—1916	1917—1978	overall
PCIM(t—0)	—.225 ***	—.361 ***	—.320 ***
—1	—.020	.301 **	.018
—2	.058	.091	.074 **
—3	.051 *	.004	.079 **
—4	.074 **	.109	.099 ***
—5	.065 **	.208 *	.104 ***
—6	.051 *	.035	.055
—7	.017	.025	.028
PCTFR(t)	.079 **	.133	.015
	R^2 = .624	R^2 = .427	R^2 = .460

The coefficients marked with '***' are significant at the 99 % level, those with '**' at the 95 % level, and those with '*' at the 90 % level. The given values of R^2 are unadjusted, the adjusted values being 5—10 % lower.

When comparing MODEL 1 and MODEL 2 (both in Table 1) we find that for the premodern period infant mortality changes in the year before do not seem to be important in explaining current fertility changes, but for the modern period it is important, thus showing the same structural change we had identified before. For the

earlier period, however, and for both periods together infant mortality lagged by 3—5 years seems to have some significant explanatory value.

Looking at age-specific fertility rates in addition to total fertility has many advantages. First we would expect women in the prime childbearing ages to react differently with respect to replacement or infanticide than very young or older mothers would do. As, of course, infant mortality is not available by age of mother we have to assume here that increases or decreases in infant mortality happen to all age groups of mothers in a similar fashion. — Furthermore, the fertility rates of different age groups are essentially independent observations; they are not influenced by each other, although they react to certain common stimuli. Thus looking at the relationship for different age groups gives us the possibility to see if the pattern observed so far holds for these subpopulations, too.

In the Appendix Table the estimates of coefficients under MODEL 1 and MODEL 2 are given, where instead of PCTFR the variable PCFERT is used which is the annual percentage change in the fertility rate of the age group given at the top of the table.

For 1783—1916 MODEL 1 again gives a negative coefficient for lag zero (though not significant in most age groups) and a highly significant positive coefficient for fertility of the previous year which is strongest in the prime childbearing ages. MODEL 2 leads for most age groups to significant negative coefficients at lag zero and for the older age groups even at lag one. This could mean that the fertility depressing effect of a mortality crisis lasts longer for older women due to lower fecundability or a higher probability of remaining widowed than for younger women. — There is no clear positive effect of infant mortality on fertility visible in this premodern period, except may be a slight effect for lag 2—4. From this we could conclude that the lactational or replacement effect was not very significant at that time.

For 1917—1978, however, the same MODEL 2 shows a clear positive association between current fertility and infant mortality of the year before for ages 20—39. For 20—25 the coefficient is even higher positive than that at lag one in MODEL 1 for the same period. — This finally proves that the pattern of change from premodern to modern situations which we had identified in the previous models also holds for all individual age groups, except for women aged 15—19 and 45—49 which are extreme cases not following the general pattern of behavior for several reasons (and also show the lowest values of R^2 in all models).

A further quantitative result appears which is hard to interpret. For most age groups in both periods, but especially in 1917—1978, PCFERT(t—5) seems to have a significant effect on PCIM(t), very much in contrast to PCFERT at lags 3, 4, 6, and 7 which are not at all significant. A possible statistical explanation for this can be found by looking at the autocorrelation function for IM and PCIM where we found that lag 5 is more highly positively correlated to the current value than lag two. We suspect that the reason for this lies in the way the annual rates were generated, which probably involved 5-year interpolation of the denominators as mentioned before.

Last but not least it is worth noting that for both periods together MODEL 2 gives highly significant positive coefficients at lags 2—6, especially in the older age groups. There seems to be no immediate answer as to the significance of those variables because this was not the case for any of the two periods separately. When, however, comparing the corresponding coefficients for the periods 1783—1916 and 1917—1978 we find that those in 1917—78 are much higher than those in 1783—1916 and therefore the high positive coefficients at lags 2—6 can be seen as a mainly modern phenomenon which only became significant when the premodern observations were added to those of 1917—1978.

A look at child mortality age 1—2

Since data on child mortality are available it is of interest to compare the findings from the analysis of infant mortality and fertility to an application of the same models to child mortality instead. This can help to check the importance of the measurement errors due to the definition of the infant mortality rate which also includes the deaths of infants born in the previous calendar year. Furthermore the lactational effect should be much weaker in this case.

Table 2 gives the estimated coefficients for the models where PCM1-2 is taken instead of PCIM for the premodern period 1783—1916.[3] At first sight we find that for MODEL 2 the coefficients of the change in the crude death rate simultaneously with the explained change in child mortality are extremely high. Furthermore, we see that the coefficients of fertility at lag zero are all positive. From this we can conclude that the annual fluctuations in child mortality age 1—2 must be much more similar to those in the crude death rate than the fluctuations in infant mortality were. — Still in MODEL 1 we find that the coefficients for fertility change at lag one are very high and positive. Also, in contrast to most models with infant mortality, fertility change at lag 2 is significantly positive, at least for mothers aged 15—34.

What is the meaning of these findings? As the dependent variable is mortality age 1—2, we have to assume that those children born at t-1 and t-2 are in the same cohort as those whose deaths we try to explain. Hence, the argument has to be that members of a large cohort experience high child mortality and those of a small cohort lower child mortality. In MODEL 1, Table 2, both coefficients for fertility at lag 1 and lag 2 are (significantly) positive for all age groups. These positive coefficients tell us that the possible measurement error discussed above is not important. If there were serious bias, b2 and b3 should have different signs. Secondly, the same fact implies that the path via »fertility stress» is much more important than the reversed path via truncation of lactation or replacement in that early period. This corresponds to the findings of the previous sections.

[3] For the modern period the data, unfortunately, were not available in computerized form.

Table 2. Distributed lag models for age-specific fertility rates and child mortality rate age 1—2.

Time period 1783—1916

MODEL 1 PCM1—2(t) as dependent variable

Independent variable	15—19	20—24	25—29	30—34	35—39	40—44	45—49
PCFERT(t—0)	.017	.579 **	.218	.099	.361 *	.176	.089
—1	.577 ***	.916 ***	1.011 ***	.681 ***	.665 ***	.690 ***	.558 ***
—2	.213 *	.335 *	.405 *	.504 **	.195	.216	.105
—3	—.133	—.179	—.293	—.106	—.179	—.297	—.158
—4	—.336 ***	—.551 ***	—.586 ***	—.572 **	—.666 ***	—.493 **	—.157
—5	—.034	—.111	—.217	—.362	—.187	—.171	—.166
—6	.102	.014	—.016	—.057	—.112	—.056	—.018
—7	—.071	.035	.182	—.011	.153	.231	—.110
PCDR(t)	1.417 ***	1.521 ***	1.421 ***	1.414 ***	1.503 ***	1.415 ***	1.361 ***
	R^2 = .811	R^2 = .804	R^2 = .807	R^2 = .791	R^2 = .797	R^2 = .802	R^2 = .788

MODEL 2 PCFERT(t) as dependent variable

Independent variables	15—19	20—24	25—29	30—34	35-39	40—44	45—49
PCM1—2(t—0)	—.024	.051 *	.012	—.028	.013	—.006	—.078
—1	.019	.008	.022	.036	.034	—.007	—.046
—2	—.022	—.003	—.001	.021	—.002	.051 **	—.032
—3	.024	.011	.006	.004	.025	.012	.024
—4	—.005	—.012	.003	.038 *	—.021	.031	.024
—5	.010	.001	.020	.010	.036	.019	.019
—6	—.019	.018	.006	.002	—.009	.004	.011
—7	.028	.013	.023	—.003	.017	—.021	—.008
PCDR(t)	—.323 ***	—.335 ***	—.216 ***	—.219 ***	—.308 ***	—.246 ***	—.087
	R = .385	R^2 = .551	R^2 = .491	R^2 = .429	R^2 = .391	R^2 = .427	R^2 = .114

Findings from the analysis of annual fluctuations

Various statistical models were applied to estimate the importance of different paths of causation between infant mortality and fertility (shown in Figure 1) from short-term variations in those time series. All models of analysis, i.e. cross-lagged correlation analysis for absolute values and for annual percentage changes, distributed lag regressions for total fertility and for age-specific fertility rates, and finally the consideration of child mortality age 1—2, give essentially the same result:

In premodern Finland high fertility tends to increase infant mortality in the following year, whereas the infant mortality experience of the previous years does not seem to be an important determinant of current fertility. In the modern period (after 1917), however, the structure of determination seems to be reversed and high infant mortality experience in a certain year tends to increase fertility in the following years.

In terms of the interpretative model given we can conclude that there is no evidence for a replacement strategy or for a strong lactational effect in premodern Finland; but evidence for a significant effect of 'fertility stress' and possible direct or indirect infanticide appears. As the following section will describe, this is not implausible for the premodern Finnish society. Once the practise of family limitation became accepted, there was no more need for reducing the number of living children but still some less effective involuntary factors could have been present. But the most significant change is that in modern Finland where 'fertility is within the calculus of conscious choice' there seems to be clear evidence for a replacement strategy.

To check the plausibility of assumptions made in this section and to shed more light on the determinating influence of long-term developments, we will finally focus on the impact of the secular infant mortality decline.

The secular decline in infant mortality

Official Finnish statistics give data on infant mortality rates from 1751 on. The curve of annual changes (see Figure 4) gives for premodern periods the typical picture of extremely strong fluctuations caused by famines and epidemics. There are two years of extreme cathastrophy with infant mortality rates above 300 per thousand (360.2 in 1808 and 398.2 in 1868). An interesting feature of those fluctuations is that usually peaks are not immediately followed by bottoms, but there are two or three years in between. Again, this pattern suggests that the fluctuations are not exaggerated by the possible measurement error in the infant mortality rate, but rather reflect the 'real' timing of changing mortality conditions.

While the average values of the rate show a declining trend, the amplitude of fluctuations clearly starts to diminish after 1870. This picture seems to correspond with the expectation that a society becomes increasingly resistant against epidemics and crop failure as it undergoes the process of modernization and technological innovation.

It seems reasonable to see 1860—1870 as a threshold since around that time infant mortality enters its steep decline which continues almost linearly until 1960 when the curve which approaches zero levels off. Hence the steep and unreversed decline in infant mortality started in Finland 40—50 years before that of fertility.

Does the Finnish evidence support or contradict the widespread notion that the decline in infant mortality initiatized the subsequent fertility decline? — Evidence from German provinces presented by Knodel (1974) raised doubts about this postulate of the usual description of the demographic transition because it appeared that the declines in fertility and infant mortality were more or less simultaneous, in some cases fertility even started to decline earlier. The Finnish case casts doubts on this postulate from the other side since 40—50 seem to be quite long for the assumed perception lag which should link fertility directly to infant mortality.

Further evidence against this argument is given from a closer analysis of infant mortality trends in the premodern period. Figure 5 gives the development of 5-year

Figure 4: Changes in infant mortality 1776—1976

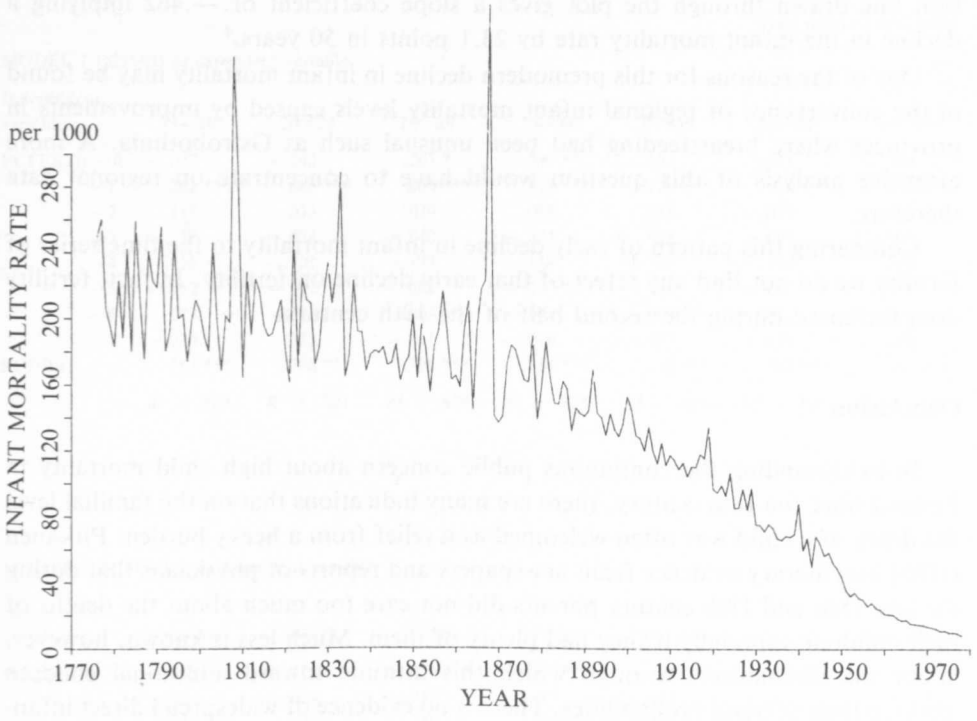
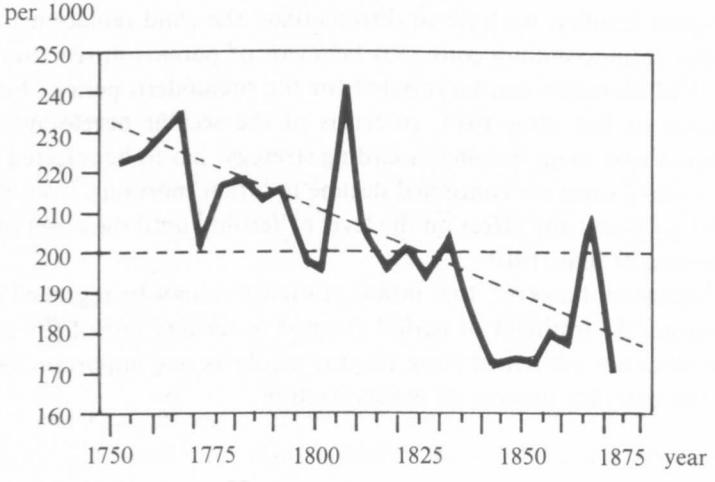


Figure 5: Infant mortality rate 1751—1875 (quinquennial data)



averages from 1751—1875 revealing significant declines even before 1880. A regression line drawn through the plot gives a slope coefficient of —.462 implying a decline in the infant mortality rate by 23.1 points in 50 years.[4]

One of the reasons for this premodern decline in infant mortality may be found in the convergence of regional infant mortality levels caused by improvements in provinces where breastfeeding had been unusual such as Ostrobothnia. A more extensive analysis of this question would have to concentrate on regional data therefore.

Comparing this pattern of early decline in infant mortality to the time series of fertility we do not find any effect of that early decline on fertility. In fact, fertility even increased during the second half of the 19th century.

Conclusion

Notwithstanding the continuous public concern about high child mortality in Finland since the 18th century, there are many indications that on the familial level the death of a child was often welcomed as a relief from a heavy burden. Pitkänen (1983) lists literary evidence from newspapers and reports of physicians that during the late 18th and 19th century parents did not care too much about the deaths of their children, especially if they had plenty of them. Much less is known, however, about the mechanisms through which this attitude toward additional children affected their survival probabilities. There is no evidence of widespread direct infanticide in Finland, but parents seemed to be quite indifferent about the health conditions of their children.

In the light of this evidence described by Pitkänen (1983), which shows that premodern child care conditions in Finland were as bad as in other European societies the findings from the statistical analysis of short-term variations which imply a dependence of infant mortality on 'fertility stress' seem even more plausible.

As to the test of the traditional child replacement hypothesis which assumes a causal link between infant mortality and fertility in a way that high infant mortality leads to higher fertility, we have to differentiate: the child replacement hypothesis in its stricter form assuming conscious behavior of parents in reaction to the incidence of child mortality can be rejected for the premodern period, but it is likely to hold more or less after 1917. In terms of the secular developments even the broader hypothesis of an 'implicit hoarding strategy' has to be rejected for the 18th and 19th century since the continued decline in infant mortality from the 18th century on did not show any effect on the level of fertility until the onset of the secular fertility decline around 1910.

The conclusion, however, that infant mortality cannot be regarded as the single most important determinant of period changes in fertility nor of the great fertility transition, does not prevent it from playing a role as one important factor among others in the complex process of modernization.

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[4] The regression line is: IM = —.462 * YEAR + 1041.

Appendix Table. Distributed lag models for age-specific fertility rates

Time period 1783—1916

MODEL 1 PCIM(t) as dependent variable

Independent variables:	15—19	20—24	25—29	30—34	35—39	40—44	45—49
PCFERT(t—0)	—.077	—.242	—.267 *	—.241 **	—.137	—.051	—.010
—1	.262 ***	.603 ***	.660 ***	.424 ***	.426 ***	.519 ***	.326 ***
—2	.118	.033	.039	.095	.011	—.029	.003
—3	—.117	—.034	—.040	—.023	—.094	—.091	—.005
—4	—.018	—.101	—.173	—.138	—.142	—.111	.014
—5	—.052	—.092	—.085	—.217 *	—.121	—.053	—.065
—6	.074	—.086	.003	—.071	—.089	—.042	.001
—7	—.039	—.083	—.011	—.048	—.077	—.017	—.008
PCDR(t)	.767 ***	.708 ***	.706 ***	.714 ***	.739 ***	.767 ***	.760 ***
	R^2 = .803	R^2 = .821	R^2 = .839	R^2 = .825	R^2 = .819	R^2 = .821	R^2 = .804

MODEL 2 PCFERT(t) as dependent variable

Independent variables	15—19	20—24	25—29	30—34	35—39	40—44	45-49
PCIM(t—0)	—.178	—.126 **	—.185 ***	—.287 ***	—.246 ***	—.449 ***	—.227 ***
—1	—.031	—.024	—.013	.015	.011	—.252 ***	—.096 **
—2	.032	.030	.049	.115 **	.042	—.046	.113 **
—3	.121 *	.064 *	.047	.046	.063	.062	.045
—4	.104	.051	.076 **	.153 ***	.016	.076	.092 **
—5	.099	.053	.067 *	.060	.078	.078	.071
—6	.059	.083 **	.055	.034	.035	.077	.053
—7	.041	.034	.041	—.017	.033	—.008	—.021
PCDR(t)	—.221 **	—.165 ***	—.088 *	—.011	—.086	.128	—.082
	R^2 = .397	R^2 = .577	R^2 = .538	R^2 = .542	R^2 = .423	R^2 = .209	R^2 = .512

Time period 1917—1978

MODEL 1 PCIM(t) as dependent variable

Independent variables:	15—19	20—24	25—29	30—34	35—39	40—44	45—49
PCFERT(t—0)	—.151	—.423 ***	—.432 ***	—.549 ***	—.597 ***	—.614 ***	—.005
—1	.059	.395 ***	.363 **	.361 **	.355 **	.420 ***	.116
—2	.011	—.038	.077	.260 *	.274 *	.194	.002
—3	—.096	.013	.067	.146	.174	.093	—.025
—4	.009	—.095	.017	—.141	—.075	.024	—.018
—5	—.208 *	—.222 *	—.341 **	—.415 **	—.384 **	—.311 *	.075
—6	.076	.095	—.038	.026	.007	.024	—.032
—7	.017	.098	.107	.190	.176	.184	.172
PCDR(t)	.370 ***	.333 ***	.325 ***	.284 ***	.288 ***	.317 ***	.384 ***
	R^2 = .285	R^2 = .513	R^2 = .512	R^2 = .563	R^2 = .564	R^2 = .546	R^2 = .233

Appendix Table cont.

MODEL 2 PCFERT(t) as dependent variable

Independent variables	15—19	20—24	25—29	30—34	35—39	40—44	45-49
PCIM(t—0)	—.237	—.337 **	—.334 ***	—.397 ***	—.413 ***	—.370 ***	—.213
—1	.116	.311 **	.374 ***	.336 ***	.257 ***	.201 *	—.152
—2	.325	.105	.088	.069	.125	.105	.245
—3	—.005	—.058	—.006	.021	.068	.083	.231
—4	—.116	.084	.096	.174	.163	.166	.214
—5	.161	.236 *	.217 *	.236 *	.221 *	.237 **	.227
—6	.244	.079	.008	.018	.064	.117	—.008
—7	.123	.060	.024	.003	.012	.052	—.119
PCDR(t)	.132	.136	104	.112	.101	.151 *	.102
	R^2 = .112	R^2 = .339	R^2 = .419	R^2 = .458	R^2 = .418	R^2 = .393	R^2 = .102

Time period 1783—1978 (= overall)

MODEL 1 PCIM(t) as dependent variable

Independent variables	15—19	20—24	25—29	30—34	35—39	40—44	45—49
PCFERT(t—0)	—.191 ***	—.418 ***	—.376 ***	—.386 ***	—.315 ***	—.295 ***	—.028
—1	.164 ***	.509 ***	.548 ***	.417 ***	.454 ***	.523 ***	.285 ***
—2	.050	.011	.088	.175 **	.124	.064	.029
—3	—.150 **	—.076	—.054	.046	.014	—.025	—.021
—4	—.033	—.074	—.053	—.030	.030	.017	.028
—5	—.129 **	—.138 *	—.168 *	—.145 *	—.065	—.047	.005
—6	.057	.029	.014	.041	.009	.037	.076
—7	—.022	.061	.066	.075	.054	.102	.061
PCDR(t)	.677 ***	.617 ***	.624 ***	.611 ***	.617 ***	.628 ***	.701 ***
	R^2 = .694	R^2 = .748	R^2 = .755	R^2 = .752	R^2 = .741	R^2 = .739	R^2 = .685

MODEL 2 PCFERT(t) as dependent variable

Independent variables	15—19	20—24	25—29	30—34	35—39	40—44	45—49
PCIM(t—0)	—.276 ***	—.277 ***	—.295 ***	—.361 ***	—.326 ***	—.306 ***	—.298 ***
—1	—.048	—.007	.023	.057	.056	—.025	—.145 **
—2	.056	.039	.053	.114 ***	.093 **	.169 ***	.096
—3	.086	.068 *	.063 *	.076 *	.122 ***	.135 ***	.195 ***
—4	.075	.066 *	.084 **	.167 ***	.087 **	.173 ***	.205 ***
—5	.079	.081 **	.097 **	.106 **	.136 ***	.161 ***	.181 ***
—6	.055	.078 **	.046	.037	.065	.096 **	.134 **
—7	.048	.043	.044	—.003	.046	.017	.023
PCDR(t)	—.096	—.018	.012	.053	.005	.016	.066
	R^2 = .227	R^2 = .358	R^2 = .378	R^2 = .442	R^2 = .382	R^2 = .398	R^2 = .147

References

Chowdhury, A. & Khan, A. R. & Chen, L. (1978). Experience in Pakistan and Bangladesh. In: The effects of infant and child mortality on fertility (ed. S. Preston), pp. 113—133.

Coale, Ansley. (1979). The demographic transition. In: UN, the population debate, dimensions and perspectives. Papers of the World Population Conference, Bucharest 1974, pp. 347—355.

Davis, Kingsley. (1945). The world demographic transition. Annals of the American Academy of Political and Social Science 237: 1—11.

Frisch, Rose. (1974). Demographic implications of the biological determinants of female fecundity. Research Paper No. 6. Cambridge, Mass: Harvard Center for Population Studies.

van Ginneken, Jeroen. (1974). Prolonged breastfeeding as a birth spacing method. Studies in Family Planning 5: 201—206.

Heer, D. (1966). Economic development and fertility. Demography 3: 423—444.

Hotelling, H. & Hotelling, F. (1931). Causes of birth rate fluctuations. Journal of the American Statistical Association 26: 135—149.

Kenny, David. (1979). Correlation and causality. New York.

Knodel, John. (1968). Infant mortality and fertility in three Bavarian villages: An analysis of family histories from the 19th century. Population Studies 22: 297—318.

—. (1978). European populations in the past: Family-level relations. In: The effects of infant and child mortality on fertility. (ed. S. Preston), pp. 21—45.

—. & van de Walle, E. (1967). Breastfeeding, fertility and infant mortality: An analysis of some early German data. Population Studies 21: 109: 131.

—. & van de Walle, E. (1979). Lessons from the past: Policy implications of historical fertility studies. Population and Development Review 5: 217—246.

Lee, Ronald D. (1975). Natural fertility, population cycles, and the spectral analysis of births and marriages. Journal of the American Statistical Association 87: 581—607.

—. (1977). Introduction. In: Population patterns in the past (ed. R. Lee). New York.

—. (1981). Short-term variation: vital rates, prices, and weather. In: The population history of England, 1541—1871; a reconstruction (ed. Wrigley & Schofield), pp. 356—400.

Mathiessen, Poul & McCann, J. (1978). The role of mortality in the European fertility transition: Aggregate level relations. In: The effects of infant and child mortality on fertility (ed. S. Preston), pp. 47—68.

Pitkänen, Kari. (1980). Registering people in a changing society — the case of Finland. Yearbook of Population Research in Finland, Vol. XVIII, pp. 60—79.

—. (1983). Infant mortality decline in a changing society. Yearbook of Population Research in Finland, Vol. XXI, pp. 46—74.

Preston, Samuel H. (1978). Introduction. In: The effects of infant and child mortality on fertility (ed. S. Preston), pp. 1—18.

Preston, Samuel H. (1980). Causes and consequences of mortality declines in less developed countries during the twentieth century. In: Population and economic change in developing countries (ed. R. Easterlin), pp. 289—340.

Scrimshaw, Susan (1978). Infant mortality and behavior in the regulation of family size. Population and Development Review 4: 383—402.

Strömmer, Aarno. (1969). Väestöllinen muuntuminen Suomessa (summary: The demographic transition in Finland). Tornio.

Turpeinen, Oiva. (1979). Fertility and mortality in Finland since 1750. Population Studies 33: 101—114.

Wappäus, J. E. (1861). Allgemeine Bevölkerungsstatistik. Leipzig.

Wray, J. D. (1971). Population pressure on families: Family size and child spacing. Chapter 11. In: Rapid Population Growth. Princeton.