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Cross-Sectional Methods for Estimating the Replacement of Infant Deaths

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BY

RANDALL J. OLSEN

I. Introduction

Demographic transition theory views a decline in infant mortality as a precondition for a decline in fertility. The transition is easily seen in time series data, but hypotheses about the nature of the transition center on the behavior of individuals, so the validation of these hypotheses is most direct using family level data. This paper describes some new methods which can be applied to cross-sectional data on families to investigate a central issue in transition theory--the replacement hypothesis.

The term replacement is often used to describe the process by which higher mortality is translated into higher fertility, although there is a variety of channels through which replacement may run. First, there is direct replacement. This term is used to describe a conscious action by a couple to increase the number of children born in response to the actual death of one of their children. In order for direct replacement to exist, the couple must have preferences over the number of children that survive

and the will and ability to alter the timing and number of births in order to move toward their fertility goal.

The second channel of replacement is hoarding. Whereas direct replacement refers to actions taken in response to actual deaths in furtherance of a couple's fertility goals, hoarding refers to actions taken in response to anticipated deaths. When I use the term hoarding it involves differential actions by couples responding to the different mortality rates they face. A third replacement channel related to hoarding is societal replacement. This refers to customs of a culture which arise in response to a common level of mortality. For example, taboos against intercourse during religious festivals may be practiced to enable the society to attain a level of fertility which generates a reasonably stable population. If this activity is common to everyone in a sample I will not be able to detect it with the cross-sectional methods I employ below. However, if differential societal replacement exists at the village level, and my data contains individuals from many villages, it may emerge as hoarding in my estimates.

Fourth, there is biological replacment which arises because for physiological reasons the death of a child shortens the interval to the next birth. If breastfeeding prolongs the period of sterility after birth, then when a nursling dies this period of sterility will be shortened, mimicking direct replacement.

The objective of this paper is to show how the effects

of direct replacement, hoarding, and biological replacement can be separated and then estimated. The focus will be on the methodology for doing this although data from Maylaysia will be used to illustrate the method. Some of the methods employed use statistical techniques more fully described elsewhere. Only a brief description of these techniques will be provided here. The final estimate for the rate of replacement in the Maylaysian data is somewhere above thirty to forty per cent, with the biological effect via breastfeeding only accounting for about twelve per cent. Replacement is greatest for children who die soon after birth with the attempt to replace being concentrated early in the birth interval. This timing of replacemant makes it difficult to separate from the effects of breastfeeding. There is also an indication in the data that breastfeeding is used as a means of contraception.

II. Methods of Data Analysis

In this paper I will consider analytical methods which are applied to family level data. This is a logical place to start since aggregate cross country and time series specifications should follow from the family level specification.

There are two basic methods used to investigate replacement with the great diversity in empirical work being

generated by variations on these basic themes.

First, there are the parity progression methods which take the data on births and deaths for a family and seek to explain the timing and frequency of births using the timing and frequency of deaths as well as other variables. Those who use the data in this way either focus on parity progression (closure of a birth interval) as influenced by mortality, or on the length of birth intervals. Second, there are the methods which seek to explain the number of births in a family as a function of either the number of deaths or the death rate. No matter what the basic approach taken a number of complications await the empirical investigator, such as: 1) How does one separate the biological effect of breastfeeding from the behavioral direct replacement response? 2) How is one's response to 1) affected if breastfeeding is consciously used to control fertility? 3) How does one separate the direct response to a death (direct replacement) from responses to anticipated deaths (fertility hoarding)? 4) Families that desire more children tend to have more children but having more children leaves them exposed to a greater probability of suffering an infant death. Has this been taken into account? 5) What about other observable biological or socio-economic determinants of fertility and mortality? 6) Is it possible that unobserveable factors influence both mortality and desired fertility giving rise to spurious relationships in the data?

As the methods below are developed I will try to point out their strengths and weaknesses. I will examine the Maylaysian data using one method focusing on the relation between family size and mortality and a second method using birth interval analysis.

A. Family Size Regressions

Because the central issue in the replacement literature is the effect of the death of a child on fertility, it would seem the most direct strategy would be to regress the number of births in a family on the number of deaths, that is, estimate the regression equation

$$n_i = a + rd_i + u_i$$

where n_i is the number of births in family i, d_i is the number of child deaths in the family, and u_i is the unexplained error in the regression. Unfortunately, the coefficient on deaths, r will not approximate the true rate of replacement.

The simple regression of births on deaths above will produce a misleading value of the coefficient r for two reasons. First, even if families do not follow a replacement strategy, families with more births will tend to have more deaths simply because they have more children at risk. This will produce a positive coefficient on deaths

unrelated to replacement. Second, there is substantial variation across families in the probability a child will be dead at the time the survey is conducted. Moreover, the family's infant mortality rate tends to be correlated with its fertility rate. This second factor is less well recognized, but must be taken into account when studying replacement.

In an earlier article (Olsen[1980]) I showed how one can correct the least squares coefficient of deaths in the regression above so as to remove the bias caused by these two factors. The method was further refined in Trussell and Olsen [1981] to allow for the possibility of variation in the rate of replacement across families. The accuracy of the method was checked using simulation data from known models. The bias in the ordinary least squares coefficient r is usually large enough so that even in the absence of replacement the value of r will be about one in the typical family size regression before the corrections are made.

The corrected least squares approach provides a first step toward measuring replacement. Its estimates of the rate of replacement will be reasonably accurate so long as the true rate of replacement is not close to one. (The method is exact if the true rate of replacement is zero.) However, corrected least squares does not inform us about all the aspects of replacement, leaving two points unaddressed. First, the rate of replacement it gives

combines the effects of direct replacement and breastfeeding. Second, only the effect of direct plus biological (i.e. due to breastfeeding) replacement is estimated; there is no information on fertility hoarding given by the method. When corrected least squares is applied to the Maylaysian data the estimated rate of direct replacement (including the breastfeeding effect) is estimated to be 0.21 . When studying replacement it is very important to take into account the correlation between mortality rates and fertility rates across couples. If this correlation were ignored in the Maylaysian data, the corrected least squares estimate of replacement would be 0.62 demonstrating the importance of examining this correlation. Virtually every other study of replacement ignores this correlation which may constitute a serious omission.

It is possible that part of the positive correlation between the incidence of mortality and fertility in the Maylaysian data is due to fertility hoarding. If so, part of the difference between 0.62 and 0.21 would be due to hoarding rather than spurious contamination from an unknown source.

In order to estimate the fertility hoarding component of replacement in cross sectional data it will be necessary to relate variations in fertility across families to variations in the child mortality rate across families. The true child mortality rate for a family is not observable,

the best we can do is to observe the realized child mortality rate for a family, which only measures the true rate with error. In addition, the child mortality rates for families may differ because of actions taken by the family. For example, if a family allocates more parental time to child care one would expect it would have a lower mortality rate. If conscious actions concerning inputs of time to child care are correlated with conscious actions to have children, the family's observed mortality rate may be related to fertility not because of hoarding, but because parents who desire more children also like to spend more time with them and so suffer a lower rate of child mortality. To avoid this source of contamination it is necessary to calculate the family mortality rate net of those factors which affect child survival and are possibly subject to parental choice. To do this I estimate a model of the waiting time to the death of a child over the first ten years of life for each child in the family. The method of estimating this waiting time model is described in Olsen and Wolpin [1981], although a brief description is given in the next section. The method involves the estimation of a regression equation with the length of life of a child as the dependent variable. The regressors include variables describing the physical surroundings of the household (sanitation, source of drinking water, etc.), variables measuring the allocation of time to child care, the number and ages of other siblings, sex and birthweight of the

child, breastfeeding, and finally a family specific fixed effect. (See the Appendix and Table A). This last variable captures variations in the child mortality rate which are due to factors which do not change during the time the family's children are being raised, such as the backgrounds of the parents. If all the inputs of time and goods which contribute to child survival are included, the fixed effect captures the cross-family variation in biological (and ecological) factors influencing child survival. So long as these factors are known to the family their effect on fertility should reveal the effect of exogenous changes in the child survival rate. Once I have estimated the family specific child mortality rate, that rate can be entered into the regression equation for the number of births. My family size regression will now be

$$n_i = a + rd_i + hp_i + u_i$$

where n_i is the number of births, d_i the number of deaths and p_i the family specific child mortality component. Once the spurious correlation between d_i and u_i is estimated using the corrected least squares approach, the above fertility regression can be readily estimated. When this is done the value of r, the direct replacement component, is estimated to be 0.17 and the value of h, the hoarding response to mortality rate variation, is 0.80. The contribution of the hoarding effect to total replacement

can be approximated by dividing h by mean family size (roughly 5.85 in the Maylaysian data). Hoarding accounts for replacement of 0.14 , and so the total rate of replacement is estimated to be 0.31 , which includes the effect of shortened breastfeeding arising from the death, direct replacement of dead children and hoarding. Note that when the family specific component of mortality was included in the regression I made the explicit assumption that the family was aware of its fixed effect. This assumption is certainly false, so while the family may adjust fertility in response to its anticipated exogenous component of mortality, the exogenous component used here (the estimated fixed effect) measures the couple's anticipated component with error. In addition, the estimated fixed effect measures the true fixed effect with error. The presence of measurement error in the estimated mortality rate variable implies the hoarding coefficient is biased towards zero while the bias in the direct replacement coefficient is uncertain a priori. At this point I have an estimate of replacement due to hoarding of 0.14 , which is an underestimate due to measurement error in the child mortality rate. The estimate of replacement net of hoarding is 0.17 , which is now subject to an uncertain degree of bias because the estimated child mortality rate was entered into the regression. Moreover, the estimate of replacement does not separate behavioral (i.e. direct) replacement from biological (i.e. due to breastfeeding) replacement. In

addition, the family size regressions provide no clue as to the timing of replacement. The simplest family size regression requires only data on the number of deaths and births for each family in the sample. In order to obtain more detailed information about replacement it is necessary to use not only more detailed data but also more complex methods of analysis. The next section will involve both of these.

B. Conception Interval Analysis

While the corrected least squares method is very informative about the extent of replacement, it tells us rather little about the details of the dynamic fertility process and its determinants. The Maylaysian data has detailed information on birth intervals, breastfeeding and mortality which can be exploited to give a better understanding of replacement.

If we are to understand the nature of the replacement process we must allow for factors which change through time since the most important explanatory variables are of this type. For example, breastfeeding may be viewed as a dummy variable which changes from one to zero when a child is weaned (or dies). Likewise, when we seek to explain the impact of the death of a child it is necessary to take into account when the child dies. As an example of the difficulties which can arise when one does not have the

dates of birth and death, consider the study by Ben-Porath[1978]. His method of checking for replacement was to examine whether the death of one of the couple's first k children influenced the probability the couple would have the k+1st child. Unfortunately, he did not have access to the dates of the births and deaths. To the extent that one of the first k children died after the mother's menopause, the importance of a death in promoting more births would be understated since such a death could not possibly influence the k+1st birth.

A different problem arises when one studies the length of birth intervals as a function of the survival of the child whose birth starts the interval. It is often observed that, on average, the death of a child at an early age shortens the interval considerably. Less clear is whether this reflects replacement or the effect of mortality shortening the duration of breastfeeding which in turn shortens the period of sterility following a birth. Deaths of children after age one shorten the interval to the next birth far less. This may seem to indicate that the observed shortening for early deaths is mostly an amenorrhea effect since after a year breastfeeding has a smaller effect upon fecundability. However, such a conclusion is not warranted. When an older child dies the interval to the next conception may already be closed, so even vigorous replacement behavior in such a case would have no effect on the length of interval to the birth of the child who followed the dead

child. Even a small difference in the mean birth interval for deaths after age one versus no death may be evidence such couples are pursuing a replacement strategy.

One of the main objectives of this paper is to separate the biological effects of breastfeeding from the behavioral effects of replacement. There is room for disagreement over the issue whether couples choose the extent of breastfeeding in part to limit births. If they do, part of the biological effect of breastfeeding must be counted as behavioral. This point has been made by Schultz[1976]. Even if couples do not choose breastfeeding to control fertility, there is still a problem in estimating the impact of breastfeeding on fertility. The difficulty arises because the death of a nursling marks the end of the contraceptive effect of breastfeeding as well as the possible beginning of replacement behavior. Most child deaths occur early in life when there will be substantial uncertainty over over whether the child had been weaned. Since most data sets do not include detailed data on how long individual children are breastfed, one cannot determine which deaths interrupted breastfeeding and which didn't. It will be easy to confound the effects of breastfeeding and replacement, so to estimate either effect requires both to be accurately estimated. It is on this rock that most studies of replacement and/or the contraceptive effects of breastfeeding founder. Because they did not have data on the duration of breastfeeding, Knodel[1968] and Chowdhury et. al.[1978] tried to infer the

effect of mortality by examining the interval between birth i and birth i+1 as a function of the deaths of children at parity i-1 and earlier. They viewed the shortening of the interval from i to i+1 following the death of the ith child as being hopelessly contaminated by the breastfeeding effect. Their strategy is certainly conservative, but if (as appears to be the case below) the behavioral replacement response is immediate and strong in the current interval and only very mild in subsequent intervals, this approach will eliminate all but the Cheshire cat's smile from the data.

It should be pointed out that the waiting time from marriage to the first conception is important information since this interval reflects fecundability in the absence of breastfeeding. Because most mothers in Maylaysia breastfeed their children for at least a few months, the interval to the first birth provides the best information about fecundability in the absence of breastfeeding. A natural question in this connection is whether couples have lower fecundability immediately following marriage since the woman is likely to be young. A comparatively long first interval might reflect adolescent subfecundity rather than a small ability of breastfeeding to lengthen birth intervals.

The well known rapid rise in natality rates moving from women under 20 to women 20-24 is either eliminated or reversed when one looks at age specific natality for married women (see United Nations[1976]). The sharpness of the rise

in certain developed countries certainly suggests this may be due to pregnancy inducing marriage. Such induced marriages reduce the mean interval from the date of marriage to the first birth. This will lead to an overstatement of the ability of lactation to reduce birth intervals and, concomitantly, to an understatement of the effect of direct replacement. To prevent extremely young women from entering the sample, no first interval was begun before age fifteen.

To facilitate the empirical work I will deal with the interval between a birth and the next conception leading to a birth rather than the interval between births. Let t be the length of time for the jth mother between birth $i-1$ and the date of conception for the ith child, where if $i-1$ equals zero the date of marriage is used as the start of the interval. The probability density function for the length of the interval is assumed to be

$$f(t_{ij}) = X_{ij}\theta + Z_{ij}(t_{ij}) + v_j \qquad (1)$$

where $Z_{ij}(t_{ij})$ represents exogenous variables whose values change through time with the j subscript indexing couples and the i subscript indexing intervals. The X_{ij} are variables whose values do not change through time, and v_j is a fixed effect specific to the couple. The particular form of the probability density function in (1) is chosen because it allows fixed effects in waiting time models to be estimated. The full details of the method can be found in

Olsen and Wolpin[1981]. The specification in (1) generates a tractable regression model which, besides allowing time varying explanatory variables and fixed effects, also takes into account that in the analysis below the length of the intervals is subject to a fixed upper limit. The method used avoids the coefficient bias which results by using ordinary least squares when the dependent variable is truncated (Pearson and Lee [1908]). Both open and closed birth intervals may be present in the data, so virtually all the most difficult complications of waiting time models can be taken into account with a fraction of the computational effort which maximum likelihood imposes.

Unless one of the regressors which changes over time is elapsed time, the specification in (1) implies that the probability density function is uniform over time. The implicit assumption of a uniform density can be relaxed by using elapsed time as an explanatory variable in (1). Table 1 gives the sample relative frequencies for the length of birth intervals when the potential length of the birth interval is five years or longer. I only consider the woman's reproductive history up to age 45 or the date of the survey, so the potential length of some intervals is restricted. For the first two years the assumption of a level density function is fairly good, so below I analyze the data by considering the first twenty-four months of each interval with the woman being considered sterile for the first two months following a live birth. All birth

intervals which are longer than twenty-four months are used in the second step of the analysis which looks at the elapsed time from the twenty-fourth month of the interval to a birth. Only the second twenty four months are considered in the second step so that only replacement behavior during the first four years of an interval will be studied. While this may result in an understatement of the true replacement effect, Table 1 shows 22.4% of all spells are longer than four years. Since there are 5.85 births per family on average, 14.6% of the intervals will be final open intervals. Therefore fertility after four years is a little under 8%.

One of the advantages of the specification in (1) is that it has a family specific fixed effect which is assumed to affect the probability density function for waiting time for a particular couple in the same way for all intervals. This is an important modification since it allows the investigatior to control for differences in both the observed and unobserved characteristics of the family which are fixed through time. The fixed effect may capture much of the variation in fecundability and desired fertility across couples. In addition, the fixed effect will capture variation in the family specific mortality rates which will eliminate the effect of hoarding on the length of the birth interval.

In Table 2 I show the effect of the mortality variables alone on the probability of a birth within the two

sets of intervals. The first mortality variable which will be used is the number of deaths which occurred prior to the birth which started the interval ("Prior Deaths"). The second mortality variable is the number of deaths within the interval of children who were born no later than the birth which started the interval ("Interval Death"). This variable changes over time since a death can occur anytime in the interval, and the effect of such a death on the length of the interval depends upon when the death occurs. For example, if a child in the family dies during the two month period of sterility following a birth, then that causes the interval death variable to be one over the next 46 months. If, in addition to this death, other deaths of children born at or before the beginning of the interval occur, then the interval death variable is incremented by one at these times. Figure 1 plots the interval death variable when death occurs at months one, eight and thirty-six of the interval. In the first panel the variable starts at one (note the analysis begins after the first two months following a birth) and jumps to two at month eight. If no birth occurs by month twenty-four then the second waiting time regression examines the elapsed time from month twenty-four to a birth and the interval death variable starts out at two and jumps to three at month thirty-six. The specification in (1) implies that the earlier a death occurs in an interval, the larger its impact on the mean waiting time to a birth over the first two years of that

interval.

In calculating the replacement effects of deaths on later birth intervals, I assume each death is followed on average by three more intervals. When I calculate the biological replacement effect which results because a death interrupts breastfeeding, I assume each death reduces breastfeeding by nine months, which is slightly above the mean duration of breastfeeding during the first twenty-four months in the Maylaysian sample. I assume breastfeeding has no effect upon fecundability after twenty-four months. One disadvantage of the birth interval method is that the effect of a death on intervals must be transformed into a replacement rate. Regressions of births on deaths directly estimate the replacement rate, even though that rate represents an average over couples with different preferences, fecundabilities and in different stages of the life cycle.

Tables 2-4 present the results from estimating a waiting time to conception model. The top half of each table shows results based upon equation (1). The first column shows the effect of each explanatory variable upon the probability of a conception leading to a live birth anytime from sixty days to twenty-four months after a birth. For example, in Table 2 if a child dies during the first sixty days of its life, the probability a conception occurs from sixty days to two years after its birth rises by 0.495. If the child died at month thirteen the rise would be half

as large since the family's exposure to an interval death would be half as great. By way of contrast, if the first child dies before the second child is born, that represents a prior death which only increases the probability the third child will be conceived within twenty-four months of the second birth by 0.036. The constant term in Table 2 shows in the absence of any deaths the probability of another conception within two years is 0.411. The numbers beneath the coefficients are t-statistics.

The second column gives the same results for all intervals lasting over two years, from month twenty-four through forty-eight. For example, a neonatal death raises the probability of a successful conception between months twenty-four and forty-eight by 0.033 given no conception occurs within the first two years following a birth.

Finally, the third column combines the results of the first two and shows the effect of the explanatory variables on the waiting time to conception subject to a forty-eight month upper limit.

The bottom half of the tables calculates the replacement rates using first the increases in the probability of a conception within the first four years and second, by dividing the changes in birth intervals by the mean closed birth interval (25 months).

The most striking result in Table 2 is that replacement is strong and immediate with rather little replacement type behavior occurring after the first two

years in a birth interval. Since most infant deaths occur in the first sixty days, it appears that children that die after living for two or more years tend not to be replaced. If child deaths are more likely to occur when the couple is older, dividing by the mean birth interval will overstate replacement since the added time at risk to natality will come when birth intervals are longer. The degree of overstatement depends upon the timing of deaths and the rate at which birth intervals lengthen with age for biological reasons.

The other explanatory variables used are "Age", which is the mother's age in months at the start of the interval, and "Husband Present" which is a time varying variable which takes on the value zero when the husband is absent from the household and one otherwise. The effects shown are for the husband always present versus never present in the interval, the latter event being rare in the sample. The "Parity" variable (the parity of the child which would close the interval) must be used with care for it is essentially a lagged dependent variable. A couple's fecundability or desired fertility will be captured by parity since if a woman of a particular age has more children than average, her previous birth intervals are likely to have been short. For such a woman the best guess is that the current interval will also be short if the high parity reflects a desire or tendency to have many children. To avoid confounding the interpretation of parity, I will only use this variable in

the fixed effects regressions where desired fertility and woman specific fecundability have been (presumably) captured by the fixed effect. When fixed effects are used the parity coefficient indicates higher order birth intervals are longer, whereas without fixed effects the parity coefficient indicates the higher order intervals are shorter. This is the sort of pattern one would expect when parity is positively correlated with desired fertility, since high parity children will tend to be in families that desire many children, and the fixed effect captures desired fertility. The same problem can also arise for the breastfeeding and husband present variables. If couples use breastfeeding or separation as means of limiting family size, then couples using these measures may also be practicing other unobserved methods of contraception. As a result, the tendency of breastfeeding to reduce fertility could be overstated because it tends to occur concurrently with other contraceptive actions. The fixed effect method provides the best opportunity for isolating the true biological effect of breastfeeding on fertility.

In Table 3 the birth interval analysis is repeated, this time adding breastfeeding and other explanatory variables. The overall rate of replacement is very similar to what was found in Table 2 with the shortening of breastfeeding accounting for a rate of replacement around 15%. The high rates of replacement calculated in Tables 2 and 3 may in part be traced to two complications. First,

once more the explanatory variables may be contaminated by fecundability and desired fertility. As was the case with parity, the number of deaths which have occured prior to the interval is probably correlated with fertility since women that have experienced many infant deaths likely have had many births (and short intervals) and may be expected to again experience a shorter interval. Likewise, there may be a correlation between fertility and mortality rates which would tend to move the coefficient on Interval Death upward. This correlation could be connected to fertility hoarding in which case the mortality variables could be capturing both hoarding and direct replacement. Unfortunately, this correlation could also be due to spurious ecological effects unrelated to replacement behavior. Those familiar with Olsen[1980] may recall that the presence of such a spurious correlation was the most serious complication there as well. This same type of contamination was revealed in the work of Ben-Porath[1978] when he used a fixed effects model of a different type.

Because the fixed effect results in Table 4 control for unobservable couple specific differences in desired fertility and fecundability they are probably the most reliable results of the three birth interval analyses. Investigators have often pointed out the importance of unobservable factors which differ across couples as a possibly confounding influence in replacement studies. It is certainly worth emphisizing that the use of a couple

specific fixed effect goes about as far as it is possible to go in the direction of accounting for heterogeneity of a very general sort across couples. The fixed effect method has eliminated the fertility hoarding effect due to couple specific mortality rates. The results in Table 4 are most comparable to the family size regressions in section II-A which corrected for hoarding by including the couple specific mortality component as an additional regressor. The estimated replacement effect in Table 4 (about 0.27) is larger than the direct replacement effect obtained by the final corrected family size regression (0.17). Note, however, that the corrected family size results also contain the possibly nonbehavioral breastfeeding effect which amounts to about 12% according to Table 4 . Applying this breastfeeding effect to the corrected least squares regression we obtain a 2% behavioral replacement effect as opposed to about 15% in Table 4. To obtain the total rate of replacement for Table 4 we must add in the hoarding effect, which was found to be about 0.14 in Section II-A. The reader has no doubt noticed that I obtain different replacement estimates depending upon whether they are based on the length of the interval to the next conception, or on the probability of birth during the first four years. As I noted above, the replacement rates based upon birth intervals will tend to be too large since some child deaths occur when the mother is older and her birth intervals tend to be longer.

When we compare the biological effect of breastfeeding in Tables 3 and 4 we see a smaller effect when we control for desired fertility via the fixed effect. As mentioned above, this indicates when family specific components are not taken into account the biological breastfeeding effect becomes confounded with contraceptive measures which are employed at the time a child is nursed. This suggests that perhaps breastfeeding is used in part as a contraceptive method. The effectiveness of breastfeeding as a contraceptive is not great. Without taking into account family specific effects, nine months of breastfeeding lengthens the birth interval by about 4.7 months according to Table 3, presumably reflecting the amenorrhea effect. When fixed effects are taken into account a better estimate of the effect is about 3.8 months. In a study of Turkish women, Fisek et. al.[1981] estimated the effect of breastfeeding on amenorrhea, although without using fixed effects, and their findings predict that nine months of breastfeeding should lengthen postpartum amenorrhea (and hence the birth interval) by about 4.5 months, a result surprisingly close to Table 3. A great many investigations into the effect of breastfeeding on amenorrhea have been made; I cite the Fisek study because it employs regression analysis and readily generates predictions.

While the Maylaysian data has detailed data on breastfeeding, the conception interval analysis described above could be performed on reconstructed family histories.

Such histories have the date of marriage and the dates of birth and death for the children, but not the date of weaning. If there is information from another source on breastfeeding practices, a rough attempt can be made to separate the effects of breastfeeding and direct replacement. If the mean duration of breastfeeding is fifteen months, then the date of weaning for each child in the retrospective survey could be set at the earlier of age fifteen months or the age at death. The presence of a first interval following marriage during which breastfeeding was absent will supply sufficient sample variation in breastfeeding to approximately decompose the effects of breastfeeding and direct replacement. This method is subject to (unavoidable) bias because of the obvious error in the weaning date, but it does provide a systematic way of combining within a single framework reconstructed family histories with information on regional breastfeeding practices.

C. Parity Progression and Birth Interval Analysis

The analysis of replacement in Tables 2 - 4 centered on the effect of a death on the probability of having a birth in either months two through twenty-four or months twenty-four through forty-eight. Those results showed a death had its greatest effect in producing a birth in the first two years of the interval. Another way of looking at

the pregnancy history data is to ignore the waiting time between births and focus only on the effect of mortality on a couple's progression from parity to parity. It is useful to consider the properties of the parity progression ratio (PPR) method in replacement studies.

PPR methods are subject to two opposing sources of error. As I showed in Tables 3 and 4, the inclusion of a couple specific fixed effect greatly reduced the coefficients on the mortality variables. This suggests the couples with high desired fertility and/or high fecundability have both high mortality and high mortality rates. Since PPR studies do not take this complication into account their observed replacement effects could well be larger than the true effects.

The second source of error arises in the interpretation of PPR results. As Rutstein and Medica[1978] have pointed out, at low parities the differences in the PPR for couples with one death versus those with no deaths should be small since most families would desire more children regardless of the number of deaths. Rather than estimate replacement as of a particular parity as the proportional difference in PPRs between couples with and without an infant death it would be better to use the proportional difference in PPRs divided by the proportion of all families at that parity whose desired family size is at or below that parity. The use of the proportional difference in the PPR across all parities, as in Vallin and

Lery[1978], is likely to understate replacement, although the degree of understatement would be difficult to estimate in the absence of data both on desired family size by couple and the correlation of fertility and mortality. Since we cannot know which of these two opposite errors is larger, it is difficult to interpret PPR results.

III. Summary

In concluding, there are three major results in this paper. The first point which should be emphisized is the importance of taking into account the presence of a spurious correlation between fertility and mortality. It is interesting to note that in the family size regressions the failure to allow for a correlation between fertility and mortality led to the replacement rate being overstated by about 0.40. Once family specific fixed effects are introduced, which presumably control for family specific mortality, the estimated rate of replacement using the birth interval analysis drops by roughly the same amount.

Second, there appears to be a moderate amount of replacement in the Maylaysian data. Replacement due to fertility hoarding is somewhere above fourteen percent. The biological impact of a death via lactation adds about twelve percent to the rate of replacement. Mothers may breastfeed in part because it has a contraceptive side effect, so some of this biological effect may reflect behavior since the

length of breastfeeding is subject to choice. Finally, direct behavioral replacement is somewhere between five and fifteen percent which leaves the lower bound for the total extent of replacement somewhere between thirty and forty percent. Some of the effect I ascribe to breastfeeding may in fact be due to direct replacement. This bias arises with my method because, as I noted above, pre-marital pregnancies increase the estimated tendency of breastfeeding to prolong birth intervals and hence decrease the estimated effect of direct replacement.

Third, the interval regressions suggest that the behavioral response to a death is fairly immediate. When a death occurs, the monthly probability of a birth rises by about 0.0035 after accounting for the effects of lactation on amenorrhea. The effect of a death is confined to the interval in which it occurs; subsequent intervals seem little affected. The effect of a child death also diminishes the further into the interval the death occurs. Thus it appears children who die soon after birth are replaced to a greater extent than older children who die. Apparently replacement is more complex than just a simple attempt to achieve a goal for live children. This result seems to suggest that as a child ages the parents view a new baby as a progressively worse substitute for that child should it die.

It is very dangerous to generalize across cultures, but this apparent concentration of replacement behavior early in

a birth interval suggests some of the replacement which demographers have attributed to the effects of lactation on amenorrhea may instead be direct behavioral replacement. There are two sources for such confusion. First, replacement occurs during that part of the birth interval when breastfeeding is most commonly practiced. This makes it exceedingly easy to attribute direct replacement to breastfeeding in the absence of the data which is necessary to separate the two. Second, because the apparent biological impact of breastfeeding is reduced when we control for family specific factors, it appears those couples that desire many children breastfeed less. This amounts to indirect evidence that breastfeeding is used as a contraceptive (see Lithell[1981]).

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Appendix

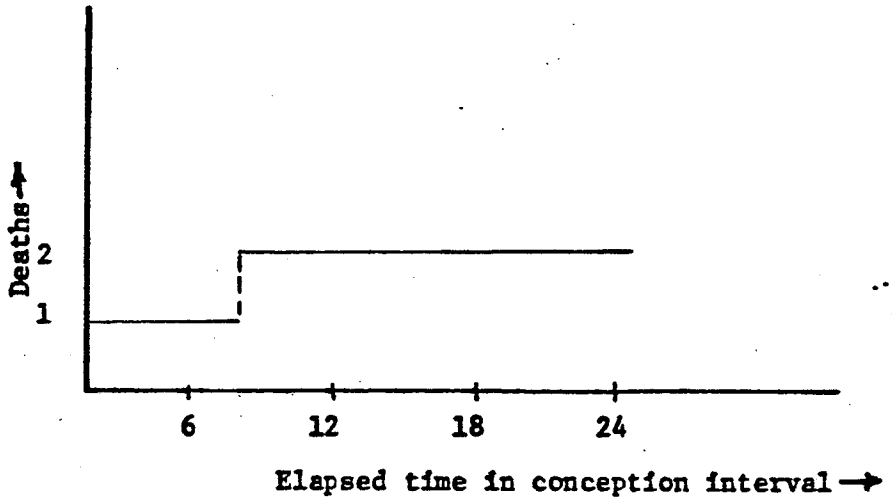
Table A gives the results for the model of length of life for children. The first column gives the coefficients when a family specific fixed effect is used, the second column shows the same specification without fixed effects. The model is estimated over the first ten years of life for each child, or the potential age at the time of the interview, whichever is smaller. As is well known, most deaths in this age range occur very early in life which means that the probability density function for length of life over the first ten years of life is not uniform but rather declines sharply with age. In order to deal with this I transformed the measure of time so that after the transformation the empirical histogram for length of life is nearly uniform up to age ten. It is not unusual for waiting time studies to transform the measure of time, most often so that the resulting data more nearly conforms to the exponential distribution. The simplest way to interpret the coefficients in Table A is to multiply by 120 to get the effect of a unit change in the variable over the first ten years of life on the probability of dying. One must be cautious when doing this since children are not breastfed for ten years, nor will they always have siblings in certain age brackets. The transformation to near uniformity I have employed implies the effects of the explanatory variables

and fixed effects are stronger at earlier ages where most of the deaths are, so that an additive model in transformed time becomes more nearly a multiplicative model in natural time units. Since the fixed effects are the elements of interest, Table A is provided only to give the reader some idea of which variables are important in determining child survival in Maylaysia.

In the presence of replacement the coefficients on the sibling variables will be biased because a death will lead a family to have more children. Since the sibling variables are essentially the average number of other children in particular age brackets during a child's potential lifetime, replacement generates causation running from deaths to the sibling variables. This confounds the interpretation of the sibling coefficients as indicating the effect of competing claims on family resources. Fortunately, the fixed effects are stochastically independent of the other coefficients so this bias is not transmitted to the fixed effects.

Figure 1

An Example of the "Interval Death" Variable



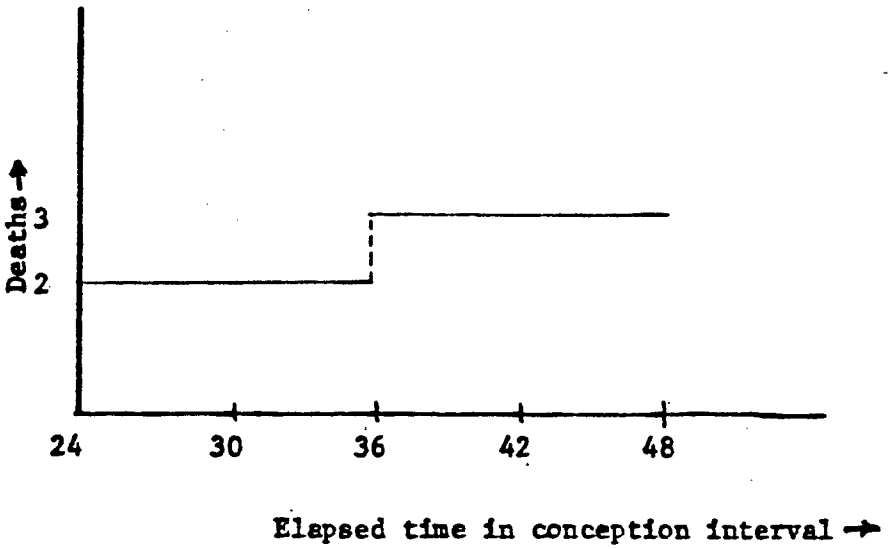


Table 1

Relative Frequency of Intervals to Conception

with Potential Length of Five or More Years

Length in Months	Relative Frequency
2–6	.129
6–12	.128
12–18	.159
18–24	.133
24–30	.100
30–36	.058
36–42	.041
42–48	.028
48–54	.022
54–60	.029
over 60	.173

TABLE 2

Conception Interval Analysis - Mortality Variables Only

Variable	Probability of Conception Within Months 2-24	Probability of Conception Within Months 24-48	Length of Interval for First 48 Months
Constant	0.411 (28.5)	0.660 (25.2)	25.60
Prior death	0.036 (1.82)	0.025 (0.69)	1.56
Interval death	0.495 (7.34)	0.033 (0.35)	-17.72

	Replacement Based on Probability of Conception During First 48 Months	Replacement Based on Length of Interval to Next Conception
Replacement over first four years	0.50	0.71
Replacement next three intervals	0.15	0.19
Total Replacement	0.65	0.90

TABLE 3

Conception Interval Analysis - Other Regressors

Variable	Probability of Conception Within Months 2-24	Probability of Conception Within Months 24-48	Length of Interval for First 48 Months
Constant	0.486 (2.27)	-0.185 (0.19)	33.21
Prior death	0.050 (2.42)	0.0382 (0.95)	-2.21
Interval death	0.328 (4.79)	0.0390 (0.41)	-11.95
Breastfeeding (9 months)	-0.113 (8.47)	—	4.69
Age Mother	0.0000585 (0.33)	-0.000239	.000821
Husband Home	0.0903 (0.42)	0.921 (0.92)	-14.21

	Replacement Based on Probability of Conception During First 48 Months	Replacement Based on Length of Interval to Next Conception
Replacement over first four years	0.34	0.48
Replacement next three intervals	0.21	0.27
Total Behavioral Replacement	0.55	0.75
Total replacement including breastfeeding	0.66	0.93

TABLE 4

Conception Interval Analysis - Fixed Effects

Variable	Probability of Conception Within Months 2-24	Probability of Conception Within Months 24-48	Length of Interval for First 48 Months
Prior death	0.00365 (0.14)	-0.0109 (0.21)	.00305
Interval death	0.0947 (1.53)	0.0678 (1.19)	-4.13
Breastfeeding (9 months)	-0.0911 (7.82)	—	3.78
Age Mother	0.00570 (15.9)	0.00230 (4.61)	-0.23
Husband Home	1.03 (3.26)	-0.657 (2.69)	-28.17
Parity	-0.234 (17.3)	-0.153 (5.75)	10.03

	Replacement Based on Probability of Conception During First 48 Months	Replacement Based on Length of Interval to Next Conception
Replacement over first four years	0.13	0.17
Replacement next three intervals	-0.001	0.00
Total Behavioral Replacement	0.12	0.17
Total replacement including breastfeeding	0.21	0.32

Table A

Probability Density Function for Length of Life in Maylaysia

Over First Ten Years of Potential Lifetime

Variable	Fixed Effects	No Fixed Effects
Constant		0.004273 (1.56)
Birth Weight	-0.00008773 (2.64)	0.0002476 (5.79)
Sex (Male = 1, Female = 2)	-.0001599 (2.25)	-0.0002171 (2.26)
Interval to Previous Child	0.000003739 (1.24)	-0.00001254 (2.38)
First Born	0.001395 (5.17)	-0.00005097 (0.18)
Second Born	0.0008561 (5.23)	0.00004377 (0.25)
Third Born	0.0005702 (4.33)	0.0003950 (2.43)
Birth Weight if Approximate	0.00003364 (0.83)	0.0002022 (5.08)
Age Mother at Birth under 25	-0.000 4177 (0.93)	-0.00 1057 (1.61)
Age Mother at Birth 25-30	-0.0005466 (1.30)	-0.001254 (1.93)
Age Mother at Birth 30-35	-0.0005443 (1.37)	-0.0007242 (1.15)
Age Mother at Birth 35-40	-0.0006826 (1.69)	-0.001398 (2.09)
Number Live Siblings Under 1	0.003460 (6.59)	0.003857 (4.89)
Number Live Siblings 1-5	0.0004645 (5.10)	0.00008664 (0.86)
Number Live Siblings 5-10	0.0002077 (3.19)	-0.00007161 (0.74)
Number Live Siblings 10-15	0.00005470 (0.72)	0.0001425 (1.40)

Variable	Fixed Effects	No Fixed Effects
Number Relatives Present Under Age 10	-0.00008681 (1.14)	-0.00009025 (2.54)
Number Relatives Present Over Age 10	0.0001094 (1.04)	0.00007252 (0.95)
Number of Grandparents Present	-0.00001147 (0.15)	0.00004058 (0.77)
Child Care by Siblings	0.0004438 (0.67)	-0.00003019 (0.03)
Child Care by Grandparents	-0.001188 (2.50)	-0.0001366 (0.28)
Child Care by Other Persons	-0.0009798 (2.10)	-0.003359 (3.03)
Electricity	-0.0002749 (1.45)	-0.001849 (6.45)
Access to Piped Water	-0.00007635 (0.36)	0.0003413 (1.62)
Access to Toilet Facilities	0.0003231 (1.30)	0.00008547 (0.91)
Number of Sleeping Rooms	0.00003040 (0.51)	-0.00005408 (0.99)
Dwelling with Modern Walls	0.00007144 (0.31)	-0.0005400 (2.79)
Time of Mother at Home and not working	-0.0001091 (0.18)	0.0004223 (1.99)
Time of Mother at Home and working	-0.001089 (1.22)	-0.00006972 (0.17)
Time of Father at Home	0.0004696 (1.15)	-0.00006781 (0.39)
Breastfeeding	-0.004153 (18.7)	-0.004144 (12.4)
Dummy for First Year of Life	0.00528 (2.29)	0.002601 (0.97)

Note: Multiply coefficients by -120 for effect on probability of death during first ten years or by -7200 for effect on length of life. For many of the time varying variables this may constitute an extrapolation considerably outside the range of observed variation.