Compensating Effects of Breastfeeding and Contraception on the Natural Rate of Increase

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December 30, 1987

This paper is an expanded and revised version of an older draft with limited circulation. Selected sections of it were presented at the Meetings of the American Statistical Association, San Francisco, June 1987. The information used here was obtained by A. Palloni and S. Millman (NIH Grant No R01HD18474) and by A. Palloni with the collaboration of J. Marcotte (NIH Grant No R01HD15982). All the calculations performed to obtain that information and the estimates presented in this paper were carried out with the support of Center Grant No HD-5876 from the National Institute for Child Health and Human Development. We thank Larry Bumpass and James Trussell for helpful comments on earlier versions of this paper. We are also grateful to John Casterline who generously shared with us alternative estimates of inhibiting effects.

I. INTRODUCTION.

By and large, the effects of family planning and health programs on the natural rate of increase of a population are evaluated by quantifying the direct impact on the variable of interest. Thus, the long-term effects of an increase in the adoption of contraception are equated to the long-term effects on the total fertility rate. Similarly, the long-term effects of child health care programs are equated with the effects on infant and child mortality. Inasmuch as programs are likely to induce indirect as well as direct changes on the rate of natural increase, these assessments will be in error.

For example, programs to reduce fertility will have an effect on the rates of infant and child mortality if the latter are responsive to changes in the pace of childbearing. Also, family planning programs may directly affect mother's health and thus enhance the health status of recently born children. In both cases the net result will be a smaller reduction in the rate of natural increase than if fertility effects were the only ones operating. Similarly, diffusion of non-traditional breastfeeding norms, leading to lower proportions of children ever breastfed and shorter durations of breastfeeding, may have offsetting effects. In the absence of any countervailing pressure, a modern pattern of breastfeeding may shorten interbirth intervals and thus increase fertility. Simultaneously, however, a reduction in the prevalence of breastfeeding may increase infant mortality directly (due to the potentially beneficial effects of breastfeeding) and indirectly (due to shorter interbirth intervals). As before, the result will be a change in the rate of natural increase of lower magnitude than would have been the case had the norm of breastfeeding been linked only to fertility or only to mortality, but not both.

With a handful of exceptions (Knodel, 1977; Gwatkin, 1984; Trussell and Pebley, 1984), demographers and planners have paid little attention to the quantification of *net* effects to be attributed to fertility limitation or health programs. This neglect is due both to the subtle and intricate nature of the relations involved and to the paucity of information. Yet, as we show later, the magnitude of the offsetting effects is not trivial: in the cases that we examine here (Colombia, Ecuador and Peru) more than one-fourth of the fertility-induced increase in the natural rate of increase that occurs in response to adherence to non-traditional breastfeeding patterns is muted by increases in infant mortality. The compensating effects associated with increased use of contraception are somewhat weaker: increases in the use of contraception lead to net changes in the rate of increases that are between four and twenty percent smaller than they would have been if infant mortality were unrelated to birth spacing.

In this paper, we formulate a model for assessing the *potential* magnitude of the offsetting effects of changes in contraception and breastfeeding on the ultimate rate of natural increase. We use data from the World Fertility Survey for Colombia, Ecuador and Peru, three Latin American countries representing intermediate and high fertility and mortality levels in the continent.

In Section II we introduce a simple but quite general model to represent the relations between fertility, infant mortality and policy variables. In Section III we develop procedures for the estimation of the parameters of the model and propose an algorithm to calculate total fertility rates and infant mortality rates that are consistent with those parameters. In Section IV we examine the results for Colombia, Ecuador and Peru.

II. POPULATION POLICIES, FERTILITY, MORTALITY AND NATURAL INCREASE.

We focus on population policies designed to increase the proportion of women using contraception at some point during their childbearing period. Such policies are usually part of more comprehensive programs involving child and maternal care, and are not infrequently accompanied by the diffusion of new values about family sizes and practices of childrearing. The most important among the latter are those affecting breastfeeding norms (Gwatkin, 1984). The relations are shown in Figure 1. The figure displays two sets of paths. One involves the effects of programs on contraception, maternal mortality and infant mortality. The other shows the relations between breastfeeding, contraception and infant mortality. Dark lines represent paths which we estimate in this paper. Dashed lines represent paths that are not identifiable with the information available to us.

a.) The relations between programs, fertility and mortality.

The fertility effects of contraception are straightforward. By averting undesired pregnancies it has a direct effect on total fertility. Similarly, since fecundity declines with age, use of contraception to delay the occurrence of a next pregnancy has a direct effect on total fertility. The magnitude of these effects depends on the efficiency of the method of contraception used as well as on the timing and regularity of its use.

A feature of many family planning and health programs is the supply of means to improve maternal health through an increased access to health personnel or facilities. Use of these may lead to direct beneficial effect on maternal health. There may also be indirect effects. In fact, since frequent and closely spaced pregnancies have deleterious health effects, the adoption of contraceptive practice can lead to improvements in maternal health. In both cases the net outcome will be a reduction on maternal mortality (Chen et al., 1977; Trussell and Pebley, 1984).

Infant mortality can be affected either directly or indirectly. The direct effects are a consequence of expansion of medical services and increased access to and use of vaccines and medications. The indirect effects operate through maternal health and through pace of childbearing. Mothers in ill health are less able to carry a normal pregnancy to term and are more prone to give birth to underweight babies who will experience higher rates of neonatal deaths (Winikoff, 1983). Thus, improvements in maternal health should result in a change in infant mortality even if the patterns of childbearing prevailing prior to the appearance of improvements were to persist. Mortality effects that result from increased use of contraception operate in several ways. First, in most developing countries births averted by contraception would have on average been exposed to higher mortality conditions. Births averted would have been of lower birth weight, higher parities and born to very young or relatively old mothers. Second, use of efficient contraception leads to different spacing of births, with a decreased incidence of shortly spaced pregnancies. Short birth intervals appear to have deleterious health effects for both the children whose birth opens the interval and for those whose birth closes it. These effects persist after a series of confounding factors (birth order, mother's age at birth, education, mortality of previous sibling, and breastfeeding) are properly accounted for (Hobcraft, MacDonald and Rutstein, 1983; Trussell and Hammerslough, 1983; Holland, 1983; Palloni and Millman, 1986; Palloni

and Tienda, 1986; Cleland and Sathar, 1983). However, the belief in the infant survival benefits of adoption of contraception is not universal (Bongaarts, 1987).

These relations imply that a simultaneous reduction in fertility and a decrease in infant mortality should occur even if the only result of a family planning program was increased use of efficient contraception. Thus, the long-term *net* effect on the rate of natural increase should be less than what would be expected on the basis of fertility effects alone. The offsetting effects due to mortality reductions will depend on the extent and pattern of increase in the use of contraception, the strength of the relation between spacing and infant mortality, and the initial levels of infant mortality.

b.) The relations between programs, breastfeeding, fertility and mortality.

Although variable according to the social setting, the evidence available suggests that the mortality reducing effects of breastfeeding are quite substantial (path c in figure 1). These effects persist after controlling for confounding factors such as education of the mother, parity, spacing of births, mother's age at the birth of the child, and pre-existing health condition of the child (Palloni and Millman, 1986; Palloni and Tienda, 1986; Holland, 1983; Cleland and Sathar, 1983; Montgomery, Richards and Braun, 1986; DaVanzo, Butz and Habicht, 1983; DaVanzo and Habicht, 1986; Butz, Habicht and DaVanzo, 1984; Habicht, DaVanzo and Butz, 1986). Similarly, there is evidence supporting the hypothesis that the practice of breastfeeding lengthens the post-partum anovulatory period, thus reducing the probabilities of conception (path d) (Knodel, 1977; Bongaarts and Potter, 1983; Bumpass, Rindfuss and Palmore, 1986; Palloni, 1984). As a consequence, breastfeeding has direct effects on fertility and mortality that lead to changes in the natural rate of growth bearing opposite signs: a wholesale abandonment of the traditional norm of breastfeeding will lead, *ceteris paribus*, to an increase in infant mortality and to an increase in fertility. The net impact on the rate of growth will depend on the strength of the corresponding relationships. Past research has revealed that the breastfeeding-induced changes in fertility are substantially muted by corresponding increases in mortality (paths c and e) (Knodel, 1977). However, those results were based on approximative estimation procedures and rested on a body of data not representative of contemporary developing countries. Furthermore, the effects of breastfeeding on infant mortality are not only direct but indirect as well. In fact, inasmuch as the pace of childbearing affects the levels of infant mortality and is affected by the patterns of breastfeeeding, a change in the latter will produce *indirect effects* on infant mortality. A more thorough assessment of the effects of breastfeeding calls for a separation of direct and indirect effects.

c.) Estimated relations.

Although we derive estimates from more precise methods and a more adequate data base, our work is subject to several important limitations. First, we only deal with the estimation of the mediating effects *given changes in contraceptive and breastfeeding practices* (paths a and b in Figure 1). We do not attempt to understand the association between characteristics of a program and its rate of success nor do we estimate the effects that result from improvements in maternal mortality. Second, we are not able to estimate the reciprocal influences of changes in infant mortality on the pace of childbearing (path e, dashed line in Figure 1). As is known, reductions in infant mortality may lead to changes in fertility

either due to modification of family size goals or as a consequence of changes in the spacing of births that results when breastfeeding is not truncated by a death. Although it would be desirable to explicitly include such effects, we only account for them indirectly by exploring the total effects of increased contraception (as it is related to changes in family size goals) and increased duration of breastfeeding (as it is related to infant mortality). However, since our estimates apply to long-term changes, we will overestimate the offsetting effects of child mortality because we will not completely account for the total, mortality-induced reduction of fertility.

A final limitation is that we neglect the causal relation between contraception and breastfeeding. Although an almost universal and powerful negative relation exists (Millman, 1985), it is unclear just what mechanisms produce it. By treating contraception and breastfeeding as exogenous and independent of each other, we may be doing violence to the existing connection between the two. For example, it may well be the case that certain combinations of breastfeeding norms and contraceptive prevalence will be unobservable or outright impossible. However, our estimates can be taken as approximate upper limits for the ultimate effects of programs inducing changes in breastfeeding and contraception. In fact, since the relation between contraception and breastfeeding is negative, the net impact on the rate of growth attributable to each of them by a procedure that ignores their association must be considered a maximum.

The tasks accomplished in this paper can be summarized as follows: we obtain estimates of the direct effects of contraception and breastfeeding (paths a and d) to calculate the magnitude of expected changes in fertility (path b). We also obtain estimates of effects of contraception and breastfeeding that are mediated by pace of childbearing (paths (a,c) and (d,e)) and of the direct effects of breastfeeding on infant mortality (path c). Finally, we combine the total effects on fertility and infant mortality and assess the net impact on the natural rate of increase.

III. FORMALIZATION OF A MODEL TO ASSESS GROSS AND NET EFFECTS ON THE NATURAL RATE OF INCREASE

One of the most important tactical limitations of the work done in this area is the dissociation between the models that lead to an assessment of effects of fertility from those that serve to gauge the effects of mortality. Researchers who have attempted to provide quantitative estimates of program outcomes are only too aware of the problem. However, due to lack of adequate information, or a desire to maximize simplicity, they have employed approximative procedures subject to unknown margins of error (Knodel, 1977; Gwatkin, 1984; Trussell and Pebley, 1984).

Consider the following illustrative problem: suppose that one were to change conventions about breastfeeding so that a 'western-like' pattern, with low proportions breastfed and short durations prevailed. How would infant mortality and fertility change? In order to provide an answer we proceed in two stages. In a first stage we generate the joint distribution of births according to characteristics affecting mortality (birth order, age of mother at birth, length of preceding and following birth interval) that would be produced *after* the change in breastfeeding takes place. In a second stage we calculate the levels of infant mortality for a birth cohort with such a joint distribution of characteristics that experiences survivorship through the first year of life with a modern norm of breastfeeding

(low proportions ever breastfed and short typical durations). This two-stage procedure is useful to retrieve estimates of the magnitudes of all three components of change triggered by the change in breastfeeding: a direct effect on infant mortality, a direct effect on fertility (altering the pace of childbearing) and, finally, an indirect effect on infant mortality which is mediated by changes in the distribution of births by characteristics (itself a function of pace of childbearing).

A second illustration involves the estimation of effects due to the adoption of efficient contraception. It is not problematic to obtain a rough assessment of the effects on total fertility. This can be accomplished by converting a measure of change in contraceptive prevalence into a corresponding measure of reductions in total fertility. The conversion may rely on several alternative procedures to calculate the inhibiting effects of intermediate variables (Bongaarts, 1978; Hobcraft and Little, 1984; Casterline, et al., 1984; Palloni, 1984). The effects on infant mortality, however, are more difficult to obtain. The strategy that others have followed is to assume that increases in contraception result in known (imputed) changes in the pace of childbearing. The effects of the latter on infant mortality are then calculated from estimates of suitably defined covariates (Trussell and Pebley, 1984).

The procedure that we propose here eliminates the necessity of imputing changes in the pace of childbearing. Instead we estimate them directly by calculating the joint distribution of births, by characteristics relevant to infant mortality, that results from the increased use of efficient contraception. A cohort's infant mortality level is dependent on the new distribution by characteristics and the levels of infant mortality associated with each of the cells of the joint distribution.

These two illustrations can be summarized in graphic form. Figure 2 represents the steps involved in the calculation of effects and their interconnection. For simplicity we have assumed that fertility depends on background characteristics (Z) and on two intermediate variables (B, C) corresponding to breastfeeding and contraception.[1] Similarly, the force of mortality during the first year of life, $\mu(t; B, P, F, Z)$, depends on the same background characteristics, Z, and on three 'intermediate' variables (B, P, F), corresponding to breastfeeding, to the time elapsed between the birth of the index child and the birth of the previous sibling, and to the time elapsed from the birth of the index child to the following conception.[2] Since P and F are affected by both breastfeeding and contraception, $\mu(t; B, P, F, Z)$ is also ***indirectly*** affected by breastfeeding and contraception. To avoid notational cluttering we have omitted from the arguments all those factors operating indirectly. The distribution of births at some initial time, t=0, by background and intermediate characteristics is $D(t = 0; B, C, P, F, Z)$ and the total fertility rate is $TFR(B, C, Z)$. As the notation implies, the distribution of births and the total fertility rate are defined for all values of the background and intermediate variables.

The changes induced in mortality by contraception or breastfeeding follow two different paths. First, they affect $D(t = 0; B, C, P, F, Z)$. For example, an increase in contraception in a population characterized by $Z = Z_0$ and $B = B_0$ will increase the proportion of births which are preceded and followed by longer intervals and decrease the total fertility rate. Likewise, a decrease of breastfeeding will both shorten the length of the preceding and the following birth intervals, and increase the total fertility rate. Second, changes in breastfeeding affect $\mu(t; B, P, F, Z)$ directly. As suggested by the notation, we omit any direct effects that contraceptive behavior may have on mortality risks (be they genuine

or spurious effects produced by its association with unmeasured behavioral characteristics). While the effects of contraception on total infant mortality will operate only through changes in $D(t = 0; B, P, F, Z)$ alone, those associated with breastfeeding will operate through changes in $D(t = 0; B, P, F, Z)$ and $\mu(t; B, P, F, Z)$ simultaneously. Thus, the overall level of infant mortality for the cohort, $Q_0(B, C, Z)$, depends on both $D(t = 0; B, C, P, F, Z)$ and $\mu(t; B, P, F, Z)$ (see the formula at the bottom of Figure 2). [3]

To capture the relations displayed in Figure 2, we follow a two-stage procedure. First, we develop an algorithm that permits the aggregation of birth specific probabilities of conception into a distribution of births by selected characteristics, and into readily used synthetic measures of fertility. We estimate the effects of contraception and breastfeeding on those birth specific probabilities and use an aggregation algorithm to calculate the effects on the distributions of births and the total fertility rate. Second, we develop a projection routine that combines the distribution of children by relevant characteristics at time of birth (obtained in the first stage) and the estimated force of mortality throughout the first year of life. We then estimate the effects of several intermediate and background characteristics on the force of mortality and use the projection procedure to calculate their effects on infant mortality. We then assume several different regimes of breastfeeding and contraception and calculate predicted values of total fertility rate, infant mortality and natural rate of increase. These quantities are then used to assess the contributions of changes in contraception and breastfeeding to changes in the natural rate of increase.

In what follows we describe in detail each of these stages. In Part (a) we outline an approach to convert estimates of effects of covariates on the pace of each birth interval into total fertility rates and the distribution of births by selected characteristics. In part (b) we show how estimates of effects of covariates on infant mortality in combination with the distribution of births by selected variables yield estimates of the levels of infant mortality characteristic of the population.

a.) Models of fertility.

i) Formulation of the model.

The problem that we need to resolve can be stated in the following manner: given knowledge of the conditional probabilities of having a birth of order r, $(r = 1, 2, ...R)$, m months after the $(r-1)^{th}$ birth, $(m = 1, ...M)$, is it possible to convert them into synthetic measures of fertility? The answer is positive as long as we make those probabilities dependent on age of mother at the birth of the $(r-1)^{th}$ child and on age at marriage. We also show that the same procedure can be generalized to calculate the *distribution* of births by selected characteristics dependent on parameters affecting the birth spacing process.

Let us assume that the interbirth intervals can be divided into k segments, S_j, (j $1, k)$, having the pairs $(t_1(j), t_2(j))$ as boundaries. If we let $\mu^r(t)$ represent an instantaneous risk of conception, we can define

$$P^r((t_1(j), t_2(j)) \mid A_{r-1}, A_m; Z) = 1 - exp\left(\int_{t_1(j)}^{t_2(j)} \mu^r(t \mid A_{r-1}, A_m; Z)dt\right) \qquad (1)$$

as the conditional probability of having a birth of order r between $t_1(j)$ and $t_2(j)$ months after the $(r \quad 1)^{th}$ birth for a woman who had the $(r - 1)^{th}$ birth at age A_{r-1}, was married

at age A_m and is characterized by a vector of covariates Z. In what follows, this vector will include background variables such as education and residence as well as intermediate variables describing contraceptive and breastfeeding behavior. If a birth does not occur within the k segments after the birth of the previous one, we will set the conditional probability for all $t > t_2(k)$ equal to zero.

Assume further that we arbitrarily assign a birth occurring in the interval $(t_1(j), t_2(j))$ to its midpoint, so that the waiting time for the birth is equal to $t_j = .5 * (t_1(j) + t_2(j))$. [4] We can then define, $F^r(t_j \mid A_{r-1}, A_m; Z)$, the unconditional probability that a woman who had the $(r-1)^{th}$ birth at age A_{r-1}, who married at age A_m, and who is characterized by the vector Z, will have a birth at the midpoint of segment S_j:

$$F^r(t_j \mid A_{r-1}, A_m; Z) \doteq P^r(t_j \mid A_{r-1}, A_m; Z) \times \prod_{\forall t_i; i<j} (1 - P^r(t_i \mid A_{r-1}, A_m; Z)) \qquad (2)$$

where we have assumed that the following approximation holds:

$$P^r(t_j \mid A_{r-1}, A_m; Z) \doteq P^r((t_1(j), t_2(j)) \mid A_{r-1}, A_m; Z) \ .$$

The following recursive equations define probabilities of having a birth of order r, $(r = 1, R)$, conditioned only on A_m:

$$\begin{aligned}
&G^1(A_1 \mid A_m; Z) = F^1(t_j \mid A_m; Z) \ ; \ A_m + t_j = A_1 \\
&G^2(A_2 \mid A_m; Z) = \textstyle\sum_{\forall A_1} F^2(t_j \mid A_m; Z) \times G^1(A_1 \mid A_m; Z) \ ; \ A_1 + t_j = A_2 \\
&G^3(A_3 \mid A_m; Z) = \textstyle\sum_{\forall A_2} F^3(t_j \mid A_m; Z) \times G^2(A_2 \mid A_m; Z) \ ; \ A_2 + t_j = A_3 \\
&\vdots \\
&G^R(A_R \mid A_m; Z) = \textstyle\sum_{\forall A_{R-1}} F^R(t_j \mid A_m; Z) \times G^{R-1}(A_{R-1} \mid A_m; Z) \ ; \ A_{R-1} + t_j = A_R
\end{aligned} \qquad (3)$$

from which it follows that:

$$H(A \mid A_m; Z) = \sum_{r=1}^{R} F^r(A \mid A_m; Z) \qquad (4)$$

is the probability of having a birth at age A (regardless of order), and that

$$TFR(A_m; Z) = \sum_{A=0}^{A=50} H(A \mid A_m; Z) \qquad (5)$$

and

$$TFR(Z) = \int_0^\infty TFR(A_m; Z) \cdot g(A_m; Z)\, dA_m \qquad (6)$$

are, respectively, the total fertility rates for women who married at age A_m and were characterized by Z, and the total fertility rate for all women characterized by Z (regardless of age at marriage). The expression $g(A_m; Z)$ corresponds to the probability that a woman characterized by Z married at age A_m. [5]

The values of $G^r(A_r \mid A_m; Z)$ can also be used to calculate the distribution of births by characteristics. To calculate the fraction of children of order r, born to mothers aged

A_r and characterized by Z, who will be followed by a birth within x months of their own birth, we use the following expression:

$$D(t=0;r,A_r,A_m,\lambda_1;Z) = G^r(A_r \mid A_m; Z) \times F^{r+1}(x \mid A_r, A_m; Z) \tag{7}$$

where $F^{r+1}(x \mid A_r, A_m; Z)$ is obtained by interpolating between $t_j < x$ and $t_{j+1} > x$. The complement of this expression, $G^r(A_r \mid A_m; Z) - D(t=0;r,A_r,A_m,\lambda_1;Z)$ is equal to the number of children whose youngest sibling (if there is one) will be born after the x^{th} month following their birth: $D(t=0;r,A_r,A_m,\lambda_2;Z)$.

Assuming that the length of previous and following birth intervals are independent, an analogous partition of $D(t=0;r,A_r,A_m,\lambda_1;Z)$ and $D(t=0;r,A_r,A_m,\lambda_2;Z)$ yields the number of all those children of order r whose preceding sibling was born less than y months before their birth.[6] To partition $D(t=0;r,A_r,A_m,\lambda_1;Z)$ we have:

$$D(t=0;r,A_r,A_m,\lambda_1,\delta_1;Z) = D(t=0;r,A_r,A_m,\lambda_1;Z) \times \sum_{j\in R} F^r(t_j \mid A_{r-1}, A_m; Z) \tag{8}$$

where R is the set of all j's such that $t_j \leq y$. The complement of this expression, $D(t=0;r,A_r,A_m,\lambda_1;Z) - D(t=0;r,A_r,A_m,\lambda_1,\delta_1;Z)$ is equal to the number of children whose immediately preceding sibling was born before the y^{th} month prior to their birth. Note that once the quantities $D(t=0;\cdot)$ [7] are expressed as a fraction of $TFR(A_m,Z)$, we obtain the joint distribution of births by age of mother at birth of the child, by birth order, and by length of previous and following intervals. As we did before, multiplying through by the probabilities of marrying at age A_m leads to values unconditioned on the age at marriage, and dependent only on the vector of characteristics Z.

The results obtained above are now generalized in two different directions.

1.) First, we make explicit the nature of the vector Z. In this paper we will define it as follows:

$$Z = (z_1, z_2, z_3, z_4)$$

where z_1 refers to education, z_2 to residence, z_3 to contraceptive behavior and z_4 to breast-feeding behavior. Except for contraception, all these variables are treated as dichotomous indicators having values 0 and 1. Details of their definition appear in Appendix 1. In this paper our interest will center on quantities such as

$$TFR(z_1, z_2, z_3, z_4)$$

and

$$D(t=0;r,A_m,\lambda_i,\delta_j;z_1,z_2,z_3,z_4) \ .$$

2.) The second generalization relates to the removal of the implicit assumption about a constant age distribution of mothers. In order to be able to compare our results with

observed quantities derived from a cross section, we introduce weights that are proportional to a factor including attrition due to mortality and taking into account the relative sizes of successive cohorts in a growing population. These weights were selected so that they correspond to a stable population:

$$w(A_r) = e^{nA_r} \times p(A_r) \tag{9}$$

where n is the rate of natural increase and $p(A_r)$ is the probability of surviving up to age A_r exactly. Notice that we do not make $p(A_r)$ and n a function of the vector Z. Although this refinement should result in an improvement, the changes would be marginal. It was found that the weighted results were not substantially different than the unweighted ones.

The quantities $D(t = 0; \cdot)$ are then weighted by $w(A_r)$ to yield:

$$\tilde{D}(t = 0; r, A_r, \lambda_i, \delta_j; Z) = \frac{D(t = 0; r, A_r, \lambda_i, \delta_j; Z) w(A_r)}{\sum_r \sum_{A_r} \sum_{i=1}^{2} \sum_{j=1}^{2} D(t = 0; r, A_r, \lambda_i, \delta_j; Z) w(A_r)} \tag{10}$$

The above procedure permits the calculation of $D(t = 0; \cdot)$ and $TFR(\cdot)$ conditional on the vector of characteristics Z. In order to calculate estimates that apply to the most recent experience of the total population, we use weights $v(Z)$ derived from the observed distribution of last births born one to six years prior to the survey. This is preferable to a sample covering a longer interval of time since the latter is more sensitive to dating errors and may not represent recent experience. The estimates for the total population are:

$$TFR = \sum_{i=1}^{2} \sum_{j=1}^{2} \sum_{k=1}^{3} \sum_{l=1}^{2} TFR(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)}) v(z_i, z_j, z_k, z_l) \tag{11}$$

and,

$$\tilde{D}(t = 0; r, A_r, \lambda_u, \delta_w) = \tag{12}$$

$$\sum_{i=1}^{2} \sum_{j=1}^{2} \sum_{k=1}^{2} \sum_{l=1}^{2} \tilde{D}(t = 0; r, A_r, \lambda_u \delta_w; z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)}) v(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)})$$

ii) Estimation of the model.

The model described above is fully identified once we estimate the quantities

$$P^r((t_1(j), t_2(j)) \mid A_r, A_m; z) \quad .$$

In previous work we chose to use logit models to estimate the effects of background and intermediate effects. This is an acceptable choice due to two limitations of the data set and one restriction that we decided to impose. First, the data available to us is a set of reproductive histories elicited from women 15-49 at the time of a survey. This implies that the birth histories are censored and that any appropriate method of estimation employed must be of a life table type to allow correction for censoring (Rodriguez and Hobcraft, 1980; Rindfuss et al., 1982; Trussell et al., 1984; Palloni, 1984; Trussell, 1984; Bumpass, Rindfuss

and Palmore, 1986; Rindfuss, Bumpass and Palmore, 1982). Second, information on breastfeeding and contraception is only available for open and last closed birth intervals. Since the latter are representative only of the births that occurred no longer than 5 years before the survey, estimation with the entire sample of open and last closed birth intervals will induce a selection bias. As a consequence, estimation must be carried out on a subsample of all open and last closed birth intervals (Rodriguez and Hobcraft,1980; Rindfuss, Bumpass and Palmore, 1982; Palloni, 1984). Finally, we wish to impose a minimum of structure on the parameters and allow the effects of variables to be different not only by birth order but also by segments of a birth interval. Although this makes a logit model a defensible choice it is by no means the only one. We assess some alternatives in the discussion section.

Each birth interval is divided into seven segments measured in completed months since the birth of the previous child or first marriage. These segments are as follows: 0-11, 12-21, 22-31, 32-41, 42-51, 52-61, 62-120. We assume that a woman will not have a child whenever the waiting time exceeds ten years. We also assume that the effects of covariates in birth intervals of order higher than 5 are homogeneous so that the information pertaining to birth of orders 5 and higher can be pooled together. This assumption is quite realistic and resolves the problem created by the small number of cases and events observed at higher parities.

We divide the restricted sample of open and last closed birth intervals by birth order and estimate separately the following models:

$$\text{logit}(P^r((t_1(j), t_2(j)) \mid A_r, A_m; z)) = \alpha + \beta Z + \gamma(\Psi(A_{r-1} + t_1(j)) \qquad (13)$$

where α,β and γ are effects, Z is a vector of covariates and Ψ is a function. Z is a vector containing the covariates z_1, z_2, z_3, and z_4. In addition, we entered a variable to measure the effects of marital dissolution (z_5). In what follows, however, we assume no marital dissolution (the variable is set equal to zero). Two important remarks about the definition of z_3 and z_4 are in order here. First, the contrast breastfeeding/no-breastfeeding is always established 9 months before the beginning of the segment in question. This definition avoids simultaneity biases that result if longer birth intervals lead to longer breastfeeding duration rather than the other way around. (Bumpass, Rindfuss and Palmore , 1986; Palloni, 1984). Also, this contrast does not distinguish between mothers who are fully breastfeeding and those who are only partially breastfeeding. The result will be to attenuate the net effects of breastfeeding on fertility. Second, the contrast efficient contraception/inefficient contraception/no-contraception does not have a precise time locus within the birth interval. The information collected indicates whether or not the woman used contraception *sometime during the duration of the interval.* It does not distinguish between those who started out the interval using contraception, and those who used it sporadically or after a period of non-use. This will impart a downward bias to the net effects of contraception.

The coefficient γ captures the effects of age at the beginning of a segment. Since age is typically entered as a proxy for fecundity, we reasoned that a fecundity function mapped on age would be a more suitable variable than an arbitrary categorization of age. Thus we defined the function Ψ, a cubic polynomial on the proportions fecund by age estimated by Henry on a set of societies experiencing natural fertility. Throughout the paper we will refer to it as the Henry function.[8] This is not an optimum solution to the difficult problem of accounting for fecundity effects. But alternative solutions such as the use of self-reported

fecundity are even less appealing. The use of the Henry function has one clear advantage: since it is a simple function of age, it permits us to trace unequivocally the reproductive history of a woman as she moves from one birth interval to the next. It thus facilitates the task of aggregating a birth interval history into synthetic measures of fertility.

We calculate values of $P^r(\cdot)$ for any A_{r-1} A_m, Z and appropriate values for the Henry function. Notice that we calculate predicted values of $P^r(\cdot)$ ***without*** setting any of the independent variables to its mean. Thus, we are ***not*** violating any conditions imposed by the nonlinearity of the logit function. The values of $P^r(\cdot)$ thus obtained are used in the manner described in (i) above to yield predicted synthetic measures of fertility and predicted distributions of births for each one of the twenty four possible subpopulations determined by the different values of Z (See Appendix 1).

To illustrate the results of the application of the procedure to observed data, we briefly review Tables 1 and 2. These Tables are abbreviated versions of those we use since they only include information on one (not four) category of the residence-education combination and on two (not three) categories of contraceptive status. Table 1 displays the TFR for each of sixteen subpopulations in Ecuador. Reading across columns permits assessment of the influence of breastfeeding and contraception. Reading across rows supplies information about the effects of background characteristics. Table 2 shows the marginal distributions of births by parity, age of mother at birth, length of preceding interval and timing of the following conception for selected subpopulations in Ecuador. As before, reading across columns reveals the effects of contraception and breastfeeding whereas reading across rows yields information about the effects of background characteristics. It is important to highlight that the quantities that appear in Tables 1 and 2 refer to extreme regimes of contraception and breastfeeding. For example, the values in the cells 'efficient contraception, breastfeeding' correspond to a situation where all women practice efficient contraception (at all parities) and breastfeed children of all orders for at least 6 months; the cells 'no-contraception, no-breastfeeding' correspond to a situation where no women practice efficient or inefficient contraception and no women breastfeed. These regimes and associated TFR's are ideal typical in the sense that they do not reflect the actual behavior of any woman in the sample: they correspond to what would be observed if the estimated effects of contraception and breastfeeding were those observed in the sample. An estimate of the overall TFR (or of the total marginal distributions) can be obtained from a weighted average of the values in Table 1 (or Table 2). The weights must represent conditions of recent prevalence of breastfeeding and contraception. To calculate these weights we select the distribution of last births that took place between one and five years prior to the survey.

b) Model of Infant Mortality.

i) Formulation of the model.

Suppose we know that a birth cohort is distributed according to $D(t = 0; \cdot)$ and that the conditional probabilities of dying within the monthly segments 0, 1-2, 3-5 and 6-12 (in completed months) for all the possible cells contained in $D(t = 0; \cdot)$ are also known. How can we estimate the value of infant mortality for such a population? Let ${}_1q_0(r, A_r, \lambda_i, \delta_j; Z)$ be the level of neonatal mortality for children with characteristics $(r, A_r, \lambda_i, \delta_j; Z)$. Those surviving the first month of life will be:

$$D(t=1;r,A_r,\lambda_i,\delta_j;Z) = D(t=0;r,A_r,\lambda_i,\delta_j;Z) \times (1 - {}_1q_0(r,A_r,\lambda_i,\delta_j;Z)) \qquad (14)$$

The average level of neonatal mortality in the population characterized by Z will be

$${}_1\bar{q}_0 = \frac{\sum [{}_1q_0(r,A_r,\lambda_i,\delta_j;Z) \times D(t=0;r,A_r,\lambda_i,\delta_j;Z)]}{\sum D(t=0;r,A_r,\lambda_i,\delta_j;Z)} \qquad (15)$$

where the summation is over all r,A_r,λ_i,δ_j. Now let ${}_2q_1(r,A_r,\lambda_i,\delta_j;Z)$ be the value of mortality between the 1^{st} and the 3^{rd} month of life. The distribution of those surviving the second segment will be given by:

$$D(t=3;r,A_r,\lambda_i,\delta_j;Z) = D(t=1;r,A_r,\lambda_i,\delta_j;Z) \times (1 - {}_2q_1(r,A_r,\lambda_i,\delta_j;Z) \qquad (16)$$

The average level of mortality within the segment 1-3 will be

$${}_2\bar{q}_1 = \frac{\sum [{}_2q_1(r,A_r,\lambda_i,\delta_j;Z) \times D(t=1;r,A_r,\lambda_i,\delta_j;Z)]}{\sum D(t=1;r,A_r,\lambda_i,\delta_j;Z)} \qquad (17)$$

where the summation is as before. A similar segment-specific projection carried to the end of the first year of life will yield ${}_3\bar{q}_3$ and ${}_6\bar{q}_6$, the average mortality for the third and fourth segments. Chaining ${}_1\bar{q}_0$, ${}_2\bar{q}_1$, ${}_3\bar{q}_3$ and ${}_6\bar{q}_6$ together results in the value of infant mortality of the population characterized by Z.

ii) Estimation of the model.

To estimate the effects of covariates on ${}_kq_x(r,A_r,\lambda_i,\delta_j;Z)$ we use the sample of open and last closed birth intervals reported in the birth histories of the WFS. The estimated model is as follows:

$$\text{logit}({}_kq_x) = \alpha + \beta_1 Z + \beta_2 z_{3x} + \beta_3 \lambda_x + \beta_4 \delta_1 + \beta_5 \delta_2 + \beta_6 y_1 + \beta_7 y_2 \quad ; \qquad (18)$$

$$(x = 0,1,3,6;\ k = 1,2,3,6)$$

where Z is a vector of covariates containing z_1 and z_2. z_{3x} (for $x > 1$ only) is a dummy variable that assumes a value of 1 if the child was breastfed for a period of time at least as long as x.[9] Thus, it is analogous to the variable for breastfeeding in the fertility model in that it can be used to define a regime with or without breastfeeding. λ_x (see Appendix 1) is a dummy variable that assumes the value of one if the conception following the birth of the index child occurred less than three months (for x=3) and less than 6 months (for x=6) after his birth. λ_3 is used to predict mortality in the third segment (but not in the fourth) and λ_6 is used to predict mortality in the fourth segment (but not in the third). δ_1 and δ_2 are two dummy variables indicating that the length of the preceding interval is between 19 and 29 or longer than 29. y_1 is a dummy variable that assumes a value of 1 if the previous sibling died before his/her fifth birthday and before the conception of the index child. Its role is to control for mortality conditions causing a spurious relation between mortality of the index child and length of preceding interval. y_2 is the proportion of all pregnancies which ended

in fetal losses. Its role is to serve as a control for mother's health (and for the influence of the latter on children's health status). As discussed elsewhere, this model facilitates the estimation of direct as well as indirect effects of birth spacing and breastfeeding while controlling for factors causing spuriousness and eliminating simultaneous causation (Palloni and Millman, 1986; Palloni and Tienda, 1986). In particular, we believe that the estimated effects of breastfeeding may well be lower bounds.

Since our work on infant mortality was initially unconnected to our work on fertility, several inconsistencies in the definition of variables had to be resolved prior to estimation of offsetting effects. The resolution of these inconsistencies is explained in Appendix 1. Following the conciliation of definitions, we obtain predicted values of ${}_kq_x(r, A_r, \lambda_i, \delta_j; Z)$ for each cell of the distribution $D(t = 0; \cdot)$. However, in contrast to the calculations for the case of fertility, we had to rely on the simplifying assumption that the cell specific mean values of y_1 and y_2 could be used to calculate cell specific values of ${}_kq_x$ without causing unduly large distortions. There are reasons to believe that our results are robust to violation of this assumption. First, we do not use global mean values of y_1 and y_2 but instead *subpopulation specific means*. Second, we rely on our estimates of relative rather than absolute changes in mortality. As is known, relative changes in predicted values from a logit model are more robust than absolute values. This fact has been used before to provide approximate measures of policy impact (Trussell and Pebley, 1984). Further, the results that we obtain compare quite well with observed values thus lending support to our conjecture about their robustness.

For illustrative purposes Table 3 displays the predicted values of ${}_kq_x$ and the associated value of ${}_1Q_o$ in the rural subpopulations for Ecuador. The differences in mortality during the first month of life by breastfeeding status are entirely attributable to the different distribution of births by characteristics reflecting pace of childbearing. This is so because, as we indicated before, we did not estimate direct effects of breastfeeding in the first age segment to avoid biases caused by omitted variables. Similarly, the differences in mortality in all age segments by contraception status only reflect the effects of different paces of childbearing. A cursory examination of Table 3 reveals that the direct effects of breastfeeding are dominant (contrast columns 1 and 2 and 3 and 4) and that those attributable to changes in the pace of childbearing caused by contraception are somewhat weak (columns 1 and 3 and 2 and 4). Exactly analogous results apply to subpopulations with different residence and education and to the data for Colombia and Peru.

As suggested before in the case of fertility, choosing suitable weights and applying them to the values displayed in Table 3 yields an estimate of infant mortality in the population. To introduce more precision in our estimates, however, we partitioned the cases in the 'breastfeeding' cells according to duration of breastfeeding. We then calculated age-segment-specific mortality values that accounted for various breastfeeding duration. Finally, we weighted the resulting estimates of infant mortality by a proportional distribution of children by breastfeeding durations. As before, the latter was obtained from last births occurring within the period 1 to 6 years before the survey.

IV. ANALYSIS OF RESULTS.

a) The data sets.

We make use of the WFS birth histories for Colombia, Ecuador and Peru. The latter two countries are among those with the highest rates of natural increase (about .025 and .027 in 1985 respectively) and the highest prevalence of breastfeeding (the median duration of breastfeeding is around 12.28 and 8.7 months respectively) in Latin America. The current levels of infant mortality and fertility and the prevalence of breastfeeding in Colombia are lower, lying midway in a ranking of all Latin American countries. The choice of these three countries is somewhat arbitrary; however, they do permit the examination of a range of experiences in Latin America. A focus on countries with high fertility and mortality should result in the identification of a maximum bound for compensating effects. Admittedly, our estimates of compensating effects would have been different had we used African or Asian countries with higher fertility and mortality and longer breastfeeding practices. Thus, our conclusions regarding offsetting effects are necessarily restricted in scope.

The birth histories for Peru were elicited from interviews to 5640 women ever in union aged between 15 and 49. The interviews were carried out between 1977 and 1978. Those for Ecuador were obtained from 6793 women (single and ever in union) aged 15-49 in interviews carried out during 1979. Finally, the birth histories for Colombia were obtained from a sample of 5378 women (single or ever in union) aged 15 to 49 who were successfully interviewed in 1976.

b) Is the suggested procedure consistent with observables?

The accuracy of the estimated distribution of births, TFR and the levels of infant mortality that we obtain cannot be easily evaluated since we lack suitable estimates of their standard errors. In lieu of a more conventional assessment of accuracy, we use three tests to gauge the consistency of our fertility estimates and one to assess the accuracy of the estimates for infant mortality.

With the first test we compare the expected to the observed marginal distributions of births by characteristics: age of mother at birth, birth order, preceding and following intervals. Table 4 displays the results for the three countries. In columns 1, 3 and 5 we show the results obtained with our procedure and in the others we show the values observed in the sample of last and next to last births born one to six years before the survey. Although we are able to reproduce quite well the observed distributions, there are some conspicuous discrepancies. In all three cases we slightly overestimate the proportions of first order births and the proportions with a very short following conception. We also underestimate somewhat the proportions of births to very young mothers and those that were born shortly after the previous birth.[10] In no case, however, do our results deviate blatantly from the observed distributions and the potential distortions that result from the observed discrepancies are likely to be small.

The second test compares the predicted and observed values of TFR for the total population in the three countries. These are displayed in Table 5a. The figures suggest that our procedures perform with acceptable margins of errors: the maximum error occurs in Peru (7.5 per cent) and the minimum in Ecuador (1.7 per cent). These are remarkably

small errors if one considers that the simplifying assumptions made along the way are quite strong. One of them is most likely to be an important contributor to observed deviations, namely, the imposition of a unique marriage function to each of the subpopulations defined by residence and education. In fact, a comparison of TFR across these subpopulations (not shown) showed larger relative errors than those in Table 5a. But even in this more strict test the errors never exceeded 14 per cent and, more importantly, we never failed to predict the observed differentials by residence and education. A better overall predictive value can only be achieved by attaining more precision in the estimation of subpopulation-specific nuptiality functions.

The third test consists of evaluating the accuracy of predictions of the fertility inhibiting effects of contraception and breastfeeding. An inhibiting effect is defined as the proportionate reduction in the total fertility rate that is due to an intermediate variable. Bongaarts was first in suggesting a procedure to allocate inhibiting effects (Bongaarts, 1978). This procedure has been modified in a variety of ways (Casterline et al., 1984) and has been generalized to take account of individual level variation (Hobcraft and Little, 1984). The models and estimates developed here lend themselves quite naturally to calculations of inhibiting effects. As shown elsewhere, the procedure that we propose yields parity-specific and not just general inhibiting effects (Palloni, 1984). It is important for the evaluation of the net impact on rates of growth that the inhibiting effects of breastfeeding and contraception be estimated accurately.

The formulae for the calculation of the inhibiting effects are listed in Appendix 3. From these definitions it should be clear that, unlike other methods, the inhibiting effects that we calculate include the contribution of two components that are separately measured: on the one hand, there are the actual inhibiting effects within each of the subpopulations. On the other hand, we have the differential distribution of the population across the various subpopulations. The former corresponds to net inhibiting effects whereas incorporation of the latter yields gross inhibiting effects. It is to the measurement of the latter that all other methods are targeted.

To carry out the test we compare inhibiting effects due to marriage, contraception and breastfeeding. We take as baselines a set of estimates calculated using the procedure originally developed by Bongaarts and later fine-tuned by Casterline and colleagues and a set calculated utilizing a procedure suggested by Hobcraft and Little. Alternative estimates of the inhibiting effects of marriage, contraception and breastfeeding are displayed in Table 5b.

Our estimates of the inhibiting effects of marriage are always larger than those obtained by the alternative methods. The magnitude of the differences are quite significant but so are the differences between the other two alternative estimates. It is important to note that estimates of inhibiting effects of marriage have been included here for the sake of completeness but that they enter in no way in our calculations of compensating effects. Our estimates of the inhibiting effects of contraception fall right in the middle of the range established by the other two alternative estimates and the discrepancies are relatively small. The estimate of inhibiting effects of breastfeeding for Colombia is exactly the same as the average of those obtained by the alternative procedures whereas those for Ecuador and Peru are respectively smaller (by 11 percent) and higher (by 9 percent) than the average of the other two. Considering that the range established by the alternative estimates is anything

but small, our procedure produces values that are quite plausible. The exception is perhaps Peru where we almost surely underestimate the inhibiting effects of breastfeeding. In fact, the value obtained implies a median length of breastfeeding (4.5 months) which is less than half the size of the observed median duration (Ferry, 1981). This is a strong indication that, at least in Peru, we may have understated the effects of breastfeeding on pace of childbearing. Accordingly, we should expect that the magnitude of the effects on the rate of natural increase that are attributable to changes in breastfeeding will be underestimated. Similarly, the indirect effects (via pace of childbearing) that a change in breastfeeding has on infant mortality will be underestimated. These two biases operate in opposite directions: underestimating the effects of breastfeeding on fertility will bias downwards the potential increase in fertility and the corresponding increase in the natural rate of increase. However, underestimating the potential increase in infant mortality that is attributable to the indirect effects of a reduction in breastfeeding will lead to underestimating the decrease in the rate of natural increase. As a consequence, the bias in the estimate of the final increase in the rate of natural growth induced by changes in breastfeeding will not be as large as it would in the absence of indirect effects of breastfeeding on infant mortality.

The fourth test is a simple comparison of predicted and observed values of infant mortality. It should be noted that, unlike the three previous ones, this is a *total* test in the sense that it is sensitive to both the estimation of fertility and that of infant mortality. In fact, as described before, our procedure to calculate an estimate of infant mortality depends on two separate steps: first, the calculation of $D(t = 0; \cdot)$ which is dependent on the model for fertility, and second the calculation of mortality within each of the four segments of the first year of life, which is dependent on the model for infant mortality. The main results appear in Table 5c. In the case of Colombia we overestimate infant mortality by about 8 percent; in the case of Ecuador and Peru we underestimate infant mortality by about 13 and 10 per cent respectively. These are margins of error that, although exceeding a conventional ceiling of 5 per cent, are well within the range of errors routinely accepted when using indirect methods of estimation.

In summary, these tests suggest that our estimates, although not exact, are subject to acceptable margins of error. It is possible that further refinements in the input functions (such as marriage patterns) can yield further improvements. However, since our aim is to focus on *relative* rather than absolute changes, such improvements will only marginally increase the precision of estimates of compensating effects.

c) Estimating offsetting effects.

To assess the magnitude of direct and indirect effects of policies affecting contraceptive prevalence and breastfeeding norms, we define three limiting scenarios. They are designed to establish upper bounds for the magnitude of effects rather than represent historical trajectories. In the first scenario we assume complete abandonment of the breastfeeding norm but retain the prevailing pattern of contraception. This implies that children are not breastfed at all and that the practice of contraception is the same as that elicited by the birth histories. The second scenario is one where the observed norm of breastfeeding is preserved and combined with universal adoption of efficient contraception. Thus, every woman uses (at all parities) an efficient method of contraception within each of the birth intervals. This is not equivalent to a situation with no contraceptive failure or with uninterrupted use.

We merely assume that the reproductive outcomes will be those for a woman who declared having used efficient contraception in all intervals. The third scenario is a combination of the first two where cessation of breastfeeding mixes with universal adoption of contraception. As with the other two scenarios, this scenario represents a hypothetical situation. Unlike the other two, however, it incorporates simultaneously a decrease in breastfeeding and an increase in contraception. This is in accord with the negative relation observed between breastfeeding and contraception.

The outcomes of other, less extreme scenarios, can also be calculated with the procedures we suggest in this paper. However, the use of extreme cases simplifies calculations and charts the range within which the outcomes of all intermediate ones should be contained.

The characterization of each scenario preserves the composition of the population by education and residence. Only the composition by breastfeeding (or contraception) status is modified. For example, in the first scenario the proportions of cases in the 'breastfeeding' category are set to zero but the distribution by education, residence, and contraception remains unchanged.

Following the calculation of TFR and infant mortality (Q_0), we estimate the ultimate effects on the natural rate of increase applying the following stable population equality:[11]

$$NRR = e^{n \times L} \tag{19}$$

where NRR is the Net Reproduction Rate and L is Lotka's mean length of a generation. The elasticity of n relative to TFR is (approximately)

$$\frac{\partial n}{\partial TFR} \times \frac{TFR}{n} = (ln(NRR))^{-1} \tag{20}$$

whereas the elasticity of n relative to infant mortality is

$$\frac{\partial n}{\partial Q_0} \times \frac{Q_0}{n} = (ln(NRR))^{-1} \times \frac{Q_0}{(1 - Q_0)} \tag{21}$$

We assume that there are no mortality changes past the first year of life. To the extent that the effects of pace of childbearing and of breastfeeding are also significant for child mortality, the effects on the rate of natural increase that we estimate will be downwardly biased. If increases (decreases) in infant mortality have the opposite effects on child mortality because of selection by health status, our estimates of changes in the natural rate of increase will have an upward bias.

A cursory examination of these approximations reveals that in order for mortality to have perfectly offsetting effects it is necessary that its relative change be at least $(Q_0/(1 - Q_0))$ as large as the changes in TFR. Even if a population program induces the same relative changes in both infant mortality and TFR, the compensating effects of the former will mute at most a fraction of the changes induced by the latter. We anticipate then that the dominant effects will be those of fertility even when mortality changes attain the same relative magnitudes as those for fertility changes.

d) Findings.

Table 6 displays the main results for Colombia, Ecuador and Peru. The first scenario shows that in Ecuador the fertility effects of cessation of breastfeeding are highest: they

imply an increase in the TFR of 34 per cent. In Colombia and Peru these effects are less than a third of those in Ecuador (11 per cent). It is satisfying to note that the magnitude of these effects coincides almost exactly with the expected increases in fertility that are consistent with the estimated inhibiting effects (see columns C in Table 5b). The increase in infant mortality is again highest in Ecuador (122 per cent), lowest in Peru (23 per cent) and of intermediate magnitude in Colombia (80 per cent). Despite the fact that the relative changes in infant mortality are in all cases several times higher than the relative changes in fertility, the changes induced in the natural rates of increase are overpowered by changes in fertility. In Colombia one half of the increase in the rate of natural increase attributable to higher fertility is offset by the decrease induced by higher infant mortality. At the other extreme (Peru) the offsetting effects attributable to infant mortality increases are about 20 per cent. Thus, the offsetting effects induced by an overhaul of the norm of breastfeeding appear to be within a range of 20 to 50 per cent.

The results for the second scenario indicate that the fertility effects of universal adoption of efficient contraception are quite uniform: in all cases they roughly imply halving the observed TFR. These effects are larger than those implied by the estimated inhibiting effects only because in their calculation we assigned weight to women practicing inefficient contraception. Instead, our second scenario defines a situation where all women only practice efficient contraception. It is important to understand that the values of TFR in the second scenario are *not* those that would be observed if all women practiced flawless efficient contraception. Naturally, in this extreme case the corresponding value of TFR would be zero. Instead, they represent what the TFR would be if all women practiced efficient contraception with the same degree of success than those who are actually observed practicing efficient contraception in the sample. The deviations from zero in the second scenario are attributable to a) failure of contraception, b) ambiguous timing of initiation of contraception, and c) errors of response and/or modelling. We suspect that the bulk of the difference is due to the first two factors. The reductions in infant mortality that result from the resulting slower pace of childbearing are of more modest magnitude but quite heterogeneous: in no case is the decrease in infant mortality larger than 11 per cent and in Colombia it reaches a value as low as 1 per cent. It is perhaps not coincidental that the lowest reduction occurs in the country with lowest fertility, least traditional norms of breastfeeding, and lowest infant mortality. It is likely that the survivorship benefits of a more modern pace of childbearing attain a maximum at the onset of the process of adoption of contraceptive practices but subside significantly during the following stages of the process. As a consequence, the compensating effects resulting from a new pattern of contraception are lower than those obtained in our first scenario. Note that in the most favorable case (Peru) the decrease in infant mortality that follows the new contraceptive regime offsets less than three per cent of the total decrease in the rate of growth induced by a lower TFR.

The results of the third scenario are a combination of the other two with a clear dominance of the second: whereas TFR should decrease by about 50 per cent in all three countries, infant mortality should increase by as much as 109 per cent in Ecuador and as little as 10 per cent in Peru with Colombia occupying an intermediate position. The net effects on the rate of natural increase are always negative: the transition to a regime with no breastfeeding and full contraception brings about no offsetting effects. In all three cases we should expect reductions in the rate of natural increase within the range 54 to 72 per

cent.

In summary, the most important offsetting effects are those associated with a transition to a more modern breastfeeding pattern and the weakest are those that accompany the adoption of contraception. However, even in the case of complete cessation of breastfeeding, the magnitude of the offsetting changes attributable to mortality increases are no larger than 50 per cent of the total change induced by higher fertility. There are modest compensating effects when only changes in contraception occur and the mortality and fertility effects reinforce each other in a scenario where both breastfeeding and contraception patterns are revamped.

One interesting question arises regarding the composition of the offsetting effects of breastfeeding. As indicated earlier, breastfeeding affects infant mortality directly and indirectly. The direct impact is due to the beneficial effects of mother's milk on nutritional status and the immune system (Palloni and Millman, 1986). The indirect impact is due to its effects on the pace of childbearing: by prolonging the postpartum amenorrhea period, longer breastfeeding leads to longer birth intervals. Since the latter appear to have a direct effect on child survival (net of breastfeeding), it follows that a change in breastfeeding practices will also affect infant survival indirectly.

What is the relative contribution of each component? The procedures described in this paper permit the breakdown of the total effects of breastfeeding into its component parts. Let $Q_0(i,j)$ be the value of infant mortality when the distribution of births is generated by a regime of full breastfeeding, (i=1), or by a regime with no breastfeeding (i=0) and when survival through the first year is enhanced by full breastfeeding, (j=1), or reduced by no breastfeeding, (j=0). The total effects of breastfeeding can be estimated by the difference

$$Q_0(0,0) - Q_0(1,1) \tag{22}$$

The component of direct effects is estimated as

$$.5 * ((Q_0(0,0) - Q_0(0,1)) + (Q_0(1,0) - Q_0(1,1))) \tag{23}$$

and the component of indirect effects as

$$.5 * ((Q_0(0,0) - Q_0(1,0)) + (Q_0(0,1) - Q_0(1,1))) \tag{24}$$

Table 7 displays the estimated relative contribution of each component in each of three subpopulations determined by contraceptive status. Three features are worthy of note. First, as should be expected, the magnitude of the indirect effects are relatively low. Its maximum of 30 per cent is attained in Ecuador. Second, the rank according to the size of the indirect component is perfectly associated with contraceptive status: the lower the degree of efficiency of contraceptive practices, the lower the magnitude of the indirect effects. As a population increases the use of contraception it experiences a pace of childbearing that is less sensitive to changes in breastfeeding. Third, the largest magnitude of the indirect component is attained in Ecuador, the country where breastfeeding has the strongest inhibiting effects on fertility. In summary, the offsetting effects on the natural rate of increase that obtained when there is a transition from a traditional norm of breastfeeding to a more modern one are almost entirely due to the direct effects of breastfeeding on

infant mortality. The relative size of its contribution, however, is contingent on the fertility inhibiting effects of breastfeeding.

IV. Summary and Discussion

Our main purpose has been to present an integrated model to estimate the effects of two intermediate variables, contraception and breastfeeding, on fertility and infant mortality while accounting for the effects of other relevant characteristics (education and residence). This was accomplished by estimating the effects of contraception and breastfeeding on birth order-specific probabilities of conception. The latter can be aggregated to yield synthetic measures of fertility (such as the TFR) as well as the distribution of births by characteristics dependent on the implied pace of childbearing (age of mother at birth, birth order, length of previous and following interval). In addition, we estimate the effects that breastfeeding and indicators of the pace of childbearing have on infant mortality. These two separate estimation procedures can be conciliated to answer questions about the final impact that changes on contraception and breastfeeding may have on the natural rate of growth of the population.

We were particularly interested in the following three issues: **a)** if a transition from a traditional norm of breastfeeding to a more modern one takes place, both fertility and infant mortality should change. Abandonment of traditional breastfeeding should, *ceteris paribus*, lead to an increase in infant mortality and to an increase in fertility. Since these changes affect the rate of natural growth but in opposite directions, *what would be the final change that the country could experience*? Our results suggest that, at least in Latin America, as much as 50 per cent of the total increase in the rate of natural growth that would occur due to the (breastfeeding dependent) increase in fertility will be offset by increases in infant mortality. This is likely to be an upper bound for the offsetting effects, one that could occur when the levels of fertility, infant mortality and breastfeeding are high. The bulk of countries in the continent that have already started a transition in fertility and mortality and have continuously shifted toward a less traditional breastfeeding norm should experience offsetting changes of much lower magnitude. This implies that further changes in the breastfeeding norm will tend to boost the natural rate of growth without significant compensating changes attributable to infant mortality.

b) If the adoption of contraception is diffused we should expect a decrease in fertility. By and large, the introduction of contraception results in a distribution of births by characteristics that is more favorable to the survival of the infant (less birth to very young or very old mothers, lower proportions of higher order births and longer interbirth intervals). Thus, if all other factors remain constant, the diffusion of contraception should also improve infant mortality. Since both these changes affect the natural rate of growth but in opposite directions, *what would be the net change that the country would experience in the period following the adoption of modern contraception*? Our results indicate that the dominant effects are those decreasing the rate of growth, and that the compensating changes attributable to lower infant mortality are of only modest magnitude, at least at the national level.

c) Changes in breastfeeding affect infant mortality directly and indirectly. The indirect effects are dependent on the strength of the relation between breastfeeding and pace of childbearing. *What is the relative magnitude of these components*? Our results indicate

that in the Latin American context, the indirect effects can reach values as high as 20 per cent of the total effects but that these levels are unlikely in situations characterized by significant practice of contraception or where the norm of breastfeeding has shifted away from a very traditional one.

Although the results that we have obtained may apply quite well to Latin America in general, it is doubtful that they can be generalized to contexts such as those in Africa and some parts of Asia where the median lengths of breastfeeding are much longer, the practice of contraception is insignificant, and the survival conditions of infants is in general worse than those observed in Latin America. In particular, it could well be the case that both the indirect and direct effects of breastfeeding on infant mortality are higher relative to the effects on fertility. This would create an arena where the offsetting effects may be of larger magnitudes than the ones estimated in this paper.

Although the models and estimation procedure employed here do resolve important difficulties and are reasonably well suited to our purposes and data, they suffer from a variety of limitations. Some of them are unavoidable as they are inherent in the definition of the models, the estimation procedure or the interplay between the two. The solutions for others would require additional work beyond the scope of this paper. We first discuss the problems associated with estimation of fertility, then those affecting the estimation of mortality, and finally, we briefly sketch ways of relaxing some assumptions for the calculation of direct and indirect effects of breastfeeding.

a) Fertility.

First, as noted above, the calculation of distribution of births rests on an assumption about independence between the length of a previous interval and the length of the following interval. This is a simplification that runs against some empirical evidence (Cleland and Hobcraft, 1985; Braun, 1980). However, it is relatively simple to resolve if we introduce as an exogenous factor the degree of association between the two. In future analysis we plan to do so, and in addition we will test the sensitivity of the model to this particular assumption.

Second, the treatment of age within the model is not as precise as would be desirable. In fact, the assumption made about births occurring in the middle of all (except the first) segments, in combination with the definition of segments in the estimation procedures, introduces inaccuracies in the estimated age pattern of fertility. This is surmounted by adopting and using only broad definitions of age categories (say not narrower than 5 years of age). Although from the point of view of the model narrower segments would be beneficial, from the point of view of estimation this turns out to be an ill-suited tactic.

Third, although explicit in the model, the dependency on the age at marriage is *not* accounted for in the estimation procedure. The formulation of the logit model incorporates no independent effect of age at marriage except in determining the age at which the fertility process begins. This will result in errors, for example, whenever earlier marriages lead to a more rapid pace of childbearing. This has been argued for age at first birth and may well be true for age at first marriage in noncontraceptive societies. It is likely, however, that the residual effect is small. Here again, the difficulty is not unsolvable since it is always possible to include age at marriage as an additional covariate for the $P^r(\cdot)$. It should also be noted that the adoption of a single marriage function, rather than one for each

subpopulation defined by the background variables, is a source of error. It is unclear how different the results would have been had we followed a painstaking estimation process to retrieve the parameters of the marriage function for four different subpopulations (education by residence), but we suspect that it would improve estimates of differentials, but make little difference in total estimates.

Fourth, the adoption of a logit instead of a hazard model may be questioned. Although the experiments that we carried out led to very similar estimates of coefficients, the hazard model provides a more elegant way of introducing the effects of age. In fact, note that the logit model could be reformulated as follows:

$$\mu^r(t, A_{r-1}, A_m) = \mu_0^r(t)e^{\beta Z} = \mu_0^r(A_{r-1} + t)e^{\beta Z} \tag{25}$$

where, μ_0^r is a baseline hazard. The baseline hazard could then be defined as a parametric transformation of the hazard underlying the Henry function. This formulation preserves the idea of a general form for fecundity but, in addition, adjusts the underlying fecundity to the data rather than imposing a unique form, and does not lead to inconsistent values at the extremes (non-zero probabilities when the Henry function assumes a value of zero). These advantages, however, are more than outweighed by the disadvantage of having to compute the effects of multiple interaction terms in order to estimate different effects of covariates for different segments.

Finally, we make an important point about the specification of intermediate variables in the estimated logit model. Although we did estimate the effects of marital dissolution or separation, we decided to overlook its occurrence. Despite the fact that its inhibiting effects were frequently statistically significant, they applied to a very small segment of the population. Thus, while our model applies to a population experiencing no marital dissolution it can be readily modified to account for it. Also, our attempts to capture the effects of pregnancy wastage were unsuccessful probably due to faulty dating as well as outright omission of induced and spontaneous abortions. Thus, our model is valid provided that the effects of pregnancy wastage are small and unrelated to those of the intermediate variables explicitly considered here. The model also omits important intermediate variables such as frequency of intercourse, and does not explicitly consider individual heterogeneity caused by fecundity and other unmeasured individual characteristics. These omissions, in addition to the stated measurement problems of breastfeeding duration and timing of contraception, are likely to lead to understatements of the inhibiting effects of intermediate variables. A solution to this problem would require a hazard model approach incorporating heterogeneity, and would entail a formidable computational burden if the technique for aggregating the birth interval specific experience adopted here were to be preserved.

b) Mortality.

Although the model proposed for the calculation of infant mortality is quite innocent and devoid of limitations, the estimation procedure is not. Most of the limitations are rooted in the specification of the logit model itself.

First, the appropriatness of a logit model could be disputed and a call for the suitability of hazard models could be made. It has been shown elsewhere, however, that the results of the two alternative procedures are virtually identical (Palloni and Millman, 1986).

Second, and more important, there are no efficient controls for the health status of the child at birth or thereafter. This forced us to avoid altogether the estimation of breastfeeding effects in the neonatal period in order to sidestep a well known bias. At worst this omission will produce a downward bias of the *total* effects of breastfeeding on infant mortality. The estimates of breastfeeding for age segments other than the first month may have an upward bias insofar as poor health may precede and lead to curtailment of breastfeeding before entrance into an age segment and to death within it. It is difficult to anticipate what the magnitude of the offsetting biases will be. There are indications, however, that the source of maximum bias (health status at birth) leads to breastfeeding effects that are only mildly exaggerated (Montgomery, Richards, and Braun, 1986; Millman and Cooksey, 1987). We expect then that our estimates of the total effects of breastfeeding on infant mortality are downwardly biased because we choose to neglect the powerful effects of breastfeeding during the first month of life.

c) Calculation of direct and indirect effects.

The estimates of effects that we obtain rely on simplifying assumptions about contraceptive and breastfeeding status. First, the contrasts to capture contraceptive status are too coarse to reflect real situations. Ideally, we should partition the population according to the timing of contraception within birth intervals. Second, our inability to incorporate the inverse relation between breastfeeding and contraception is another important obstacle. The scenarios that we define reflect situations in which one changes without affecting the other. This may result in unrealistic representations particularly when the changes involved are extreme. Third, the distribution of births by characteristics and synthetic indicators of fertility and mortality are strictly dependent on the effects of relevant covariates estimated in the baseline models. As a consequence, the estimates of direct, indirect and offsetting effects are neither more nor less accurate than those in the baseline models. Further, they reflect the nature of the covariates themselves. For example, the estimates for the scenario with full contraception are affected by errors in response and our neglect of the timing of use of contraception in each birth interval.

Ultimately, many refinements can be introduced to resolve these and other problems. They should be introduced if what we need are more accurate forecasts of individual population parameters. The value of the model that we introduce here is that it permits us to chart the areas where further improvements will yield higher benefits in terms of the accuracy of forecasts for individual populations.

Appendix 1 : Conciliation and Definitions of Variables.

The task of calculating offsetting effects relies heavily on being able to connect the results of the aggregation of birth interval dynamics to the results of infant mortality analysis. Since initially these two tasks were performed with no intention of connecting them at all, it is natural that some inconsistencies had to be removed. In what follows we summarize the definition of variables used and, whenever needed, we explain the nature of the inconsistencies and how we attempted to eliminate them.

a) z_1 refers to education. In the analysis of mortality we distinguish three groups of women: those with no education, those with primary (complete or incomplete) and those with education higher than complete primary. In this paper we pool the first two groups so that the variable z_1 becomes a single dummy with possible values 0 and 1. In order to conciliate the mortality analysis with this definition, we calculate a mortality effect for women with $z_1 = 0$ which is equal to the weighted average of 0 (the mortality effect of those with no education) and the effect of having some primary. The distortions that this averaging may introduce are minimal since the mortality effects of having some primary education are almost indistinguishable from those of having none. We will denote the values of z_1 by $z_{1(i)}, i = 1, 2$.

b) z_2 refers to residence. In this paper and in the mortality analysis this variable is defined in identical manner ($z_2 = 1$ if rural and 2 if urban). We will denote the values of z_2 by $z_{2(j)}$, $j = 1, 2$.

c) z_3 refers to contraception; $z_3 = 1$ if a woman (or her husband) does not practice contraception during a particular birth interval, 2 if a woman practices inefficient contraception and 3 if she practices efficient contraception. We will denote the z_3 by $z_{3(k)}$, $k = 1, 3$.

d) z_4 refers to breastfeeding. Its definition varies by birth interval and segment in the analysis of fertility and by segments in the analysis of mortality. The contrast we want to establish is between a regime of no breastfeeding and one where breastfeeding is universal, and of long duration. So, unless otherwise stated, the breastfeeding variable will assume a value of 0 when there is no breastfeeding, and a value of 1 when breastfeeding is universal and lasts longer than 6 months. In some parts of the paper the definition of this variable is fine tuned to correspond better with requirements of the analysis (see Section on mortality); however, we are generally only interested in contrasting the two polar cases of no breastfeeding and maximum breastfeeding. We will denote the z_4 by $z_{4(l)}$, $l = 1, 2$.

e) λ is a variable measuring the duration between the birth of a child and the conception of the following child. In the mortality analysis it is defined as a dummy which is unity if the following conception occurred before the third month for the segment 3-6, and before the sixth month for the segment 6-12. It is undefined for the first two segments. In the fertility analysis, the time to following conception is measured in three categories: the first captures those cases where the following conception occurs

before the end of the third month after the birth of the index child; the second category represents those cases where the following conception occurs between the end of the third month and the end of the sixth month after the birth of the index child; finally, the third category represents cases where either there is no following conception or, if there is, it occurs during the period following the first six months after the birth of the index child. The definitions of λ used in fertility and mortality analyses are thus compatible.

f) δ refers to the length of preceding interval. In the analysis of mortality we use a three category definition. The first category represents cases where the birth of the index child occurs less than 20 months before the birth of the preceding child; the second category represents those cases where the birth of the index child occurs between 20 and 29 months after the birth of the preceding child; finally, the third category represents index children who are first births or those whose birth occurred longer than 29 months after the birth of the preceding child. Since in this paper we use only *two* categories (less than 20. and 20 and above) we proceed as follows: the mortality effects of the second category are combined with those of the third using a weighted average of the corresponding logistic coefficients. Here again, the distortions that result will be minimal since effects being averaged are very close to each other.

g) r refers to parity. We form three groups: birth of order one, of order two and of order three and above. The mortality and fertility analysis are consistent.

h) A_r refers to age of mother at the birth of the child. We create three categories: less than 20, between 20 and 29 and older than 29. The mortality and fertility analysis are consistent.

Appendix 2: Formulae for Inhibiting Effects

We use the following notation:

a) $V(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)})$, $(i,j,k,l = 1,2)$, is the proportion of the population in the subpopulations of education $z_{1(i)}$, residence $z_{2(j)}$, contraception $z_{3(k)}$ and breastfeeding $z_{4(l)}$ (see Appendix 1 for definitions of these variables).

b $V(z_{1(i)}, z_{2(j)}, \cdot, \cdot)$ is the proportion of the subpopulation with education $z_{1(i)}$ and residence $z_{2(j)}$.

c) $p(z_{1(i)}, z_{2(j)}, z_3 = 3)$ is the proportion of the subpopulation with education $z_{1(i)}$ and residence $z_{2(j)}$ who practice efficient contraception.

d) $p(z_{1(i)}, z_{(j)}, z_3 = 2$ is the proportion of the subpopulation with education $z_{1(i)}$ and residence $z_{2(j)}$ who practice inefficient contraception.

e) $TFR(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)})$ is the TFR for the subpopulation of education $z_{1(i)}$, residence $z_{2(j)}$, contraception $z_{3(k)}$ and breastfeeding $z_{4(l)}$.

f) $TFR_M(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)})$ is the TFR that the subpopulation of education $z_{1(i)}$, residence $z_{2(j)}$, contraception $z_{3(k)}$ and breastfeeding $z_{4(l)}$ would experience if all women married at age 15.

The inhibiting effect of marriage is:

$$I_m = \sum_{i=1}^{2}\sum_{j=1}^{2}\sum_{k=1}^{3}\sum_{l=1}^{2}\left[\frac{TFR(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)})}{TFR_M(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)})} \times V(z_{1(i)}, z_{2(j)}, z_{3(k)}, z_{4(l)})\right]$$

which is the weighted average of subpopulation specific inhibiting effects.

The inhibiting effects of contraception are defined in two stages. First, we calculate the subpopulation inhibiting effects in the presence and absence of breastfeeding; the following formulae apply in the case of efficient contraception:

$$I_{c2}(z_{1(i)}, z_{2(j)}) = \frac{TFR(z_{1(i)}, z_{2(j)}, z_3 = 3, z_4 = 2)}{TFR(z_{1(i)}, z_{2(j)}, z_3 = 1, z_4 = 2)}$$

with breastfeeding and

$$I_{c3}(z_{1(i)}, z_{2(j)}) = \frac{TFR(z_{1(i)}, z_{2(j)}, z_3 = 3, z_4 = 1)}{TFR(z_{1(i)}, z_{2(j)}, z_3 = 1, z_4 = 1)} \quad .$$

with no breastfeeding. Similar definitions apply for the inhibiting effects attributable to inefficient contraception, $II_{c2}(z_{1(i)}, z_{2(j)})$ and $II_{c3}(z_{1(i)}, z_{2(j)})$ respectively. The inhibiting effects in subpopulation $(z_{1(i)}, z_{2(j)})$ are then

$$I_c(z_{1(i)}, z_{2(j)}) = .5 \times ((I_{c2}(z_{1(i)}, z_{2(j)}) + I_{c3}(z_{1(i)}, z_{2(j)})) \times p(z_{1(i)}, z_{2(j)}, z_3 = 3) + .5 \times ((II_{c2}(z_{1(i)}, z_{2(j)}) + II_{c3}(z_{1(i)}, z_{2(j)}) \times p(z_{1(i)}, z_{2(j)}, z_3 = 2) + (1 - (p(z_{1(i)}, z_{2(j)}, z_3 = 3) + p(z_{1(i)}, z_{2(j)}, z3 = 2))$$

The overall inhibiting effect of contraception is the weighted average of these:

$$I_c = \sum_{i=1}^{2} \sum_{j=1}^{2} I_c(z_{1(i)}, z_{2(j)}) \times V(z_{1(i)}, z_{2(j)}, \cdot, \cdot)$$

Exchanging the role of breastfeeding and contraception leads to an analogous formula defining the inhibiting effects of breastfeeding.

Footnotes

1. As is the case throughout the paper, Z, B, C, F, and P may be treated as categorical indicators inducing a partition of a cohort of births. For example, Z may represent education (no education/ some education), B represents breastfeeding (breastfeeding/nobreastfeeding), C represents contraceptive status (no contraception/inefficient contraception/efficient contraception), F timing of following conception (short/long) and P length of preceding interval (short/long). In subsequent sections of the paper and to avoid unnecessary complications in the notation, the background and intermediate variables will both be included in a vector Z.

2. To simplify the notation in the example we have deleted two important determinants of infant mortality both of which are affected by the pace of childbearing: A_r, the age of the mother at the birth of the rth child and, r, the birth order of the child.

3. To simplify the figure we have assumed that the effects of variables on the force of mortality are invariant during the first year of life. This justifies the expression for infant mortality that appears at the bottom of the Figure. The assumption is relaxed throughout our calculations.

4. This assumption is necessary to take into account the age at which events occur. Unless the segments are too wide, it will not lead to large errors. For obvious reasons, the first segment has to be treated with care. Either its midpoint is around nine months or it has to be assumed that, whatever its width, all events that occur within it take place by the end of the ninth month.

5. In what follows we assume that all births occur within marriage and that the latter refer to more or less stable unions including legal marriages as well as consensual unions.

6. This partition is valid only if there is no association between length of preceding interval and length of following interval. This is a strong assumption which we are invoking for convenience. The procedure can be generalized to include situations where a known degree of correlation exists.

7. To avoid excessive notational cluttering and whenever it does not lead to ambiguities, we will abbreviate references to functions of many arguments using $(\cdot)$ or $(t;\cdot)$, where t stands for time and the dot stands for all other arguments.

8. The 'Henry function' is a set of interpolated values of proportions fecund by single years of age starting at 12. It was estimated using a cubic polynomial from ages 20 on and a logarithmic extrapolation for ages below 20. Details of the adjustment appear elsewhere (Palloni, 1984).

9. The equation for the first segment does not include a variable for breastfeeding to avoid contamination of the estimates with effects of health status at birth.

10. Understatement of births to very young mothers is probably a result of our assumption that all fertility takes place after attaining age 15. Among others, the consequence of this assumption is to slightly underestimate infant mortality.

11. The stability assumption that we make here is harmless since very similar results obtain with a rectangular age distribution. It is useful, however, to provide closed form expressions for the magnitude of relative changes that can be analyzed independently of their observed values.

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Table 1: Marginal Distributions of Selected Characteristics: Colombia, Ecuador, & Peru.

	Colombia		Ecuador		Peru	
	Expected[1]	Observed[2]	Expected[3]	Observed[2]	Expected[4]	Observed[2]
Parity						
1	.249	.223	.247	.209 (967)	.213	.179 (959)
2	.213	.189	.187	.179 (829)	.182	.163 (873)
3 +	.538	.588	.560	.612 (2828)	.606	.658 (3529)
Age						
less than 20	.095	.200	.108	.146 (673)	.066	.126 (676)
20 - 29	.483	.500	.479	.537 (2483)	.446	.515 (2762)
30 +	.423	.300	.415	.317 (1468)	.489	.359 (1923)
Length of Preceding Interval (months)						
less than 20	.176	.230	.154	.174 (805)	.189	.207 (662)
20 +	.825	.770	.847	.826 (3829)	.812	.793 (4717)
Length of Following Interval (months)						
less than 7	.098	.086	.109	.060 (281)	.128	.124 (1112)
7 +	.902	.914	.891	.939 (4353)	.872	.876 (4267)

[1] Calculated from the joint distribution D(t = o; r, Ar, γi, δj, Z) using a Coale marriage function with a = 11.06, k = .913 proportion ever married = .910 and a stable age distribution with n = .030 and $^{0}e_{0}$ = 62, United Nations Latin American pattern.

[2] Calculated from open and last closed birth intervals opened within the period 1-5 before the survey (in parentheses appear the observed number of cases).

[3] Calculated from the joint distribution D(t = o; r, Ar, γi, δj, Z) using a Coale marriage function with a = 12.75, k = .805 and proportion ever married = .937 and a stable age distribution with n = .030 and $^{0}e_{0}$ = 62, United Nations Latin American Pattern.

[4] Calculated from the joint distribution D(t = o; r, Ar, γi, δj, Z) using a Coale marriage function with a = 11.7, k = .829 and proportion ever married = .940 and a stable age distribution with n = .030, $^{0}e_{0}$ = 62, United Nations Latin American Pattern.

[5] Refers to the time elapsed from the date of the last birth to that of the current birth. The second category includes first order births.

[6] Refers to the time elapsed from the date of the current birth to that of the conception of the following. The second category includes cases with no following births.

Table 2a: Observed and Predicted Values of TFR: Colombia, Ecuador, and Peru.

	Observed[a]	Expected
Colombia	4.75	5.01
Ecuador	5.34	5.25
Peru	5.68	6.11

[a] Calculated from births by age of mother occurring during the 5 years preceding the survey.

Table 2b: Alternative Estimates of the Inhibitive Effects of Contraception, Breastfeeding and Marriage[1]

		Colombia			Ecuador			Peru		
		A	B	C	A	B	C	A	B	C
Inhibiting Effects of										
Marriage	Im	.463	.602	.706	.527	.656	.714	.503	.629	.716
Contraception	Ic	.738	.633	.676	.806	.709	.762	.847	.755	.805
Breastfeeding	Ii	.944	.846	.900	.883	.782	.748	.876	.769	.901

[1] Column A obtained using Hobcraft-Little procedure (multiplicative form, 1-36 months).
Column B obtained using a modified Bongaarts' procedure suggested by Casterline et al. (1984).
Column C obtained using our procedure.

Table 2c: Observed and Expected Values of Infant Mortality: Colombia, Ecuador, and Peru.

	Observed[1]	Expected
Colombia	.0694	.0749
Ecuador	.0760	.0671
Peru	.0960	.0877

[1] Obtained from births and deaths taking place within the first five years preceding the survey.

Table 3: Estimated Changes in Fertility, Infant Mortality and Natural Rate of Increase under Different Regimes of Contraception and Breastfeeding: Colombia, Ecuador and Peru[1]

		Colombia	Ecuador	Peru
I.	Scenario I			
	Relative Change in			
	TFR	.12	.340	.110
	Qo	.800	1.220	.230
	Relative Change in Natural Increase due to Change in			
	TFR	.170	.430	.130
	Qo	-.090	-.112	-.026
II.	Scenario II			
	Relative Change in			
	TFR	-.480	-.530	-.490
	Qo	-.012	-.040	-.110
	Relative Change in Natural Increase due to Change in			
	TFR	-.660	-.670	-.580
	Qo	.002	.004	-.013
III.	Scenario III			
	Relative Change in			
	TFR	-.470	-.450	-.450
	Qo	.780	1.090	.100
	Relative Change in Natural Increase due to Change in			
	TFR	-.640	-.570	-.530
	Qo	-.088	-.100	-.011

[1] Scenario I imposes no breastfeeding while preserving actual (observed) contraception practices.
Scenario II imposes full contraception while preserving actual (observed) breastfeeding practices.
Scenario III imposes no breastfeeding and full contraception.

Table 4: Relative contribution of the direct and indirect effects of breastfeeding on infant mortality by contraceptive behavior.

Contraceptive Behavior	Colombia			Ecuador			Peru		
	Total [a]	Indirect	Direct	Total	Indirect	Direct	Total	Indirect	Direct
No Contraception	.075	.023	.977	.104	.274	.727	.031	.066	.944
Inefficient Contraception	.075	.016	.985	.084	.200	.800	.029	.066	.944
Efficient Contraception	.075	.004	.996	.084	.191	.809	.027	.032	.969

[a] Corresponds to the total (absolute) effect of breastfeeding on infant mortality.

FIGURE 1 : REPRESENTATION OF MAIN RELATIONS

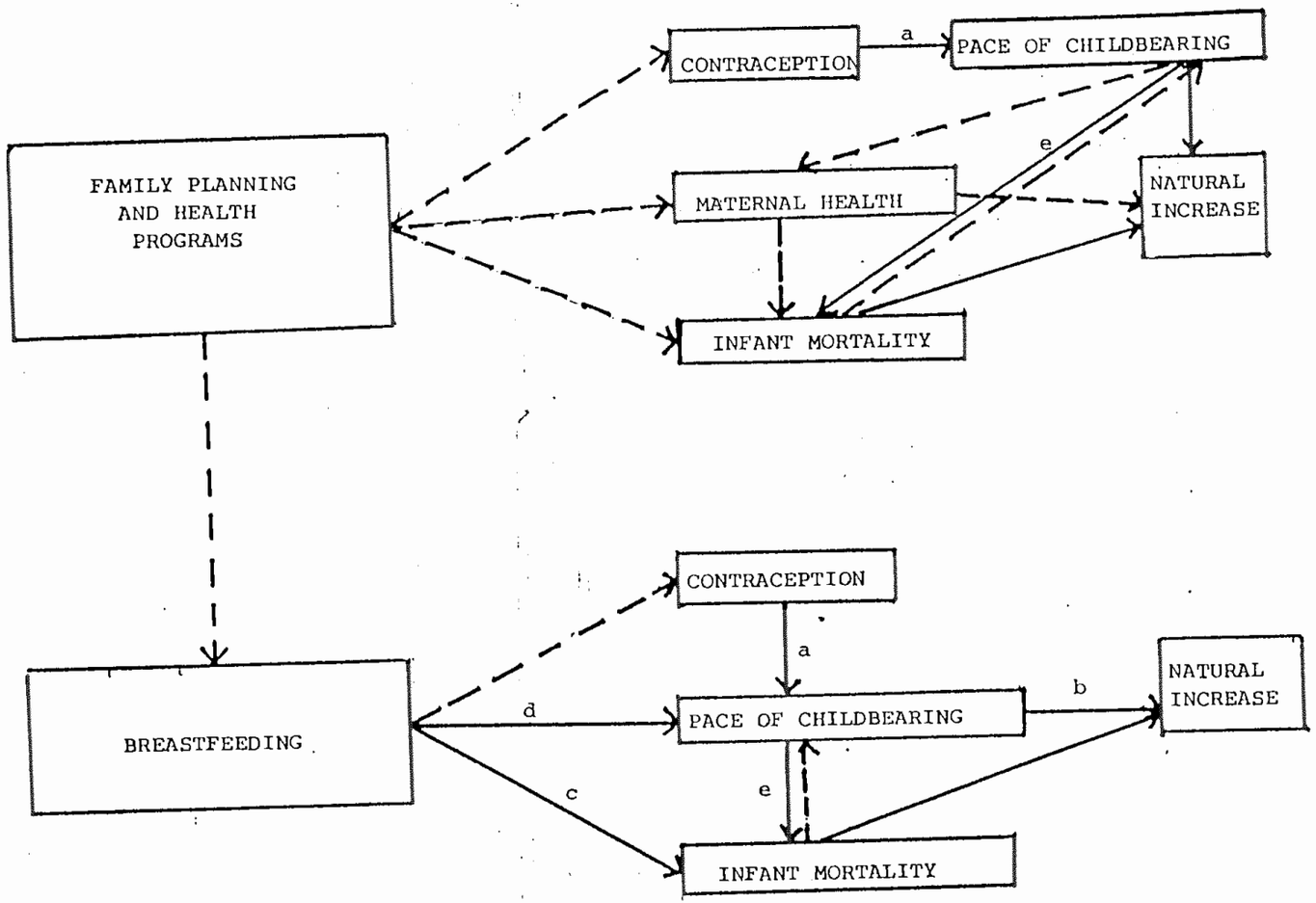
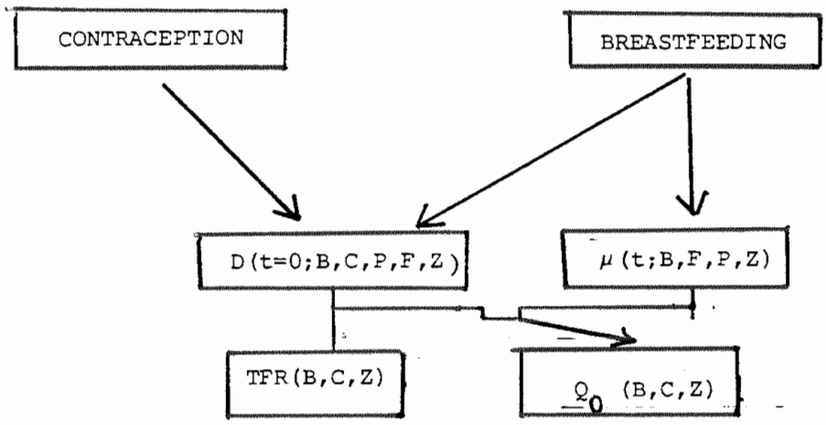


FIGURE 2 : DECOMPOSITION OF EFFECTS OF CONTRACEPTION AND BREASTFEEDING.



$$Q_0(B,C,Z) = 1 - \sum_{(B,C,F,P,Z)} \left[D(t=0;B,C,F,P,Z) \times exp\left(- \int_0^1 \mu(t;B,F,P,Z)dt \right) \right]$$

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