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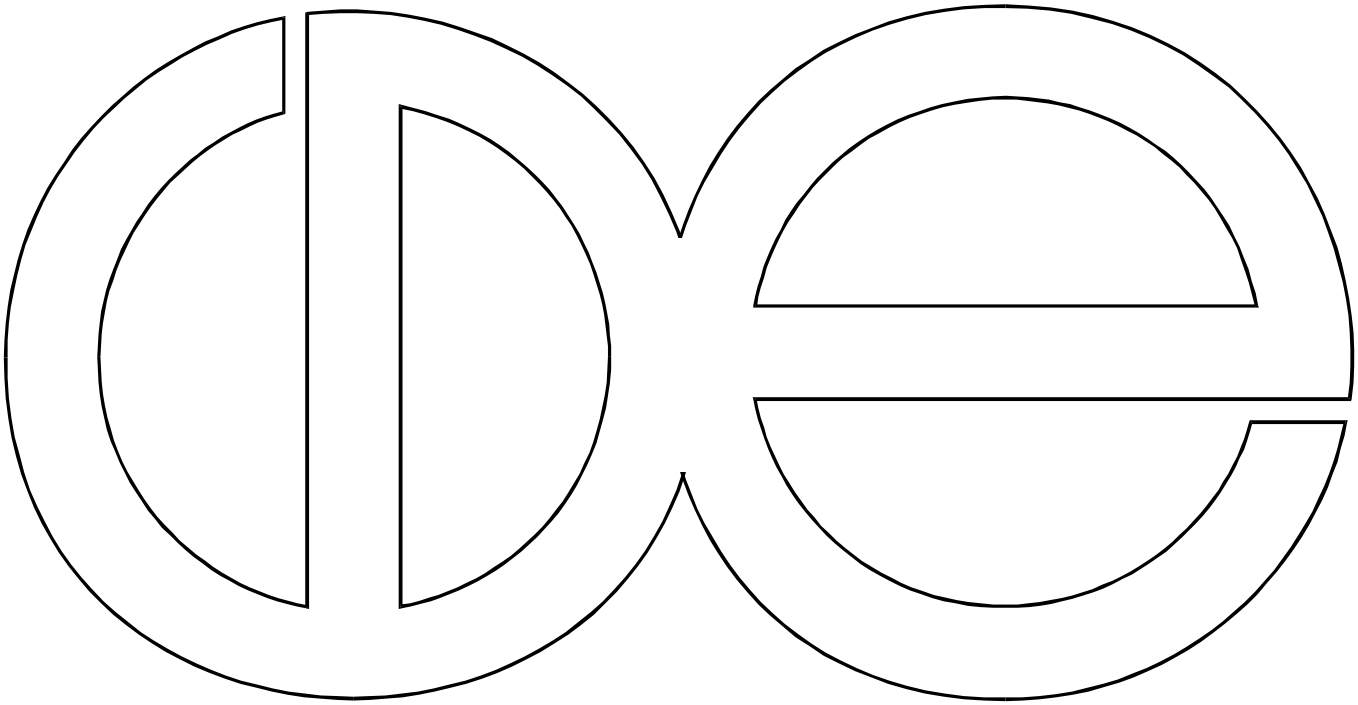

THE EFFECTS OF INFANT MORTALITY ON FERTILITY REVISITED: SOME NEW EVIDENCE

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INTRODUCTION

The relationship between infant and child mortality and fertility continues to occupy a central if somewhat unsettled place in demographic research. A recent reevaluation of the theory and evidence prepared under the auspices of the National Academy of Sciences introduces new findings and some novel and important conceptual frameworks without, however, resolving some outstanding problems and controversies (Montgomery and Cohen, 1997).

The problem is complex since it involves multiple dimensions that are difficult to disentangle empirically or even identify theoretically. On one hand, there are the effects of fertility on infant and child mortality, a relation depending mostly, but not solely, on influences of length of birth intervals and related mediating mechanisms such as breast-feeding. On the other hand, there is an effect of infant and child mortality on fertility which depends mostly on mechanisms directly associated with birth intervals. To complicate matters even more, fertility and mortality are affected by common factors which are normally unmeasured but leave indelible imprints on both processes.

In this paper we examine empirical evidence for Latin American countries during a relatively long period of time after 1920. We investigate the relation at several levels of aggregation and seek to evaluate the extent to which evidence at one level is consistent with evidence at other levels. We examine cross country information over a period of several decades, a type of data set that has been the workhorse of past research in the field. We add a detailed examination of yearly series of births, deaths, infant deaths, and socioeconomic indicators for selected countries which permit us to track the association between short-term fluctuations in fertility and infant mortality. Finally, we analyze micro level data from the Demographic and Health Surveys (DHS) and assess the relation between fertility and child mortality from individual reproductive histories. To our surprise the evidence we

assemble is quite consistent and suggests only small positive effects of child mortality on fertility, too small perhaps to impute to changes in child mortality more than modest importance as an explanatory factor of the process of fertility decline that takes place in Latin America.

The paper is organized in six sections. Section II examines the most important mechanisms producing the relation and Section III summarizes the available evidence supporting their existence. Section IV describes data from a pooled cross-section and time series for selected Latin American countries and introduces estimates of the strength of the impact of child mortality on fertility via a random effects model. Section V discusses the existence of short-term fertility responses to changes in infant and child mortality over the period 1900-1990. Section VI develops two different procedures to assess the relation between child mortality and fertility from individual data. First, we apply a technique due to Trussell and Olsen to generate a range of estimates using mothers' information on children born and surviving. Second, we use discrete hazard models on individual reproductive histories from the Demographic Health Surveys to arrive at estimates of the ultimate impact of changes in child mortality on fertility. Finally, Section VII summarizes the main results.

II. THE MECHANISMS

The effect of fertility on infant and child mortality has been thoroughly demonstrated, especially through examination of data from the World Fertility Surveys (WFS) and Demographic and Health Surveys (DHS). The results from this research provide strong evidence supporting the hypothesis that short (previous and following) birth intervals, birth order and mother's age at birth have strong effects on infant and early child mortality (Hobcraft et al., 1983; Palloni and Millman, 1986; Palloni and Tienda, 1986; Pebley and Stupp, 1987; Miller, 1989; Hobcraft, 1992). Although the actual role of alternative mechanisms underlying the relations — maternal depletion, child

competition for maternal care and household resources, lactation, crowding — has not been completely elucidated, analyses in an impressively heterogeneous array of countries reveal that the association is strong and ubiquitous and, more importantly, surprisingly robust to models, measurement procedures, and methods of estimation. These findings have potentially important policy implications which have not gone unnoticed. First, they lead to arguments according to which further mortality declines in some developing countries may not be possible unless fertility declines below current levels (Palloni, 1990; Rosero-Bixby, in press). Second, the findings sparked a yet unresolved debate about the possibility that family planning programs could ultimately have offsetting impacts on the natural rate of increase of a population as its fertility-related effects reduce the rate of increase while, simultaneously, its child mortality-related effects could potentially increase it (Trussell and Pebley, 1984; Bongaarts, 1987, 1988; Trussell, 1988; Potter, 1988; Palloni and Kephart, 1989).

But the causal relation also goes in the opposite direction since infant and early childhood mortality could affect fertility levels and patterns. The centrality of this particular relation in demographic research can hardly be exaggerated since it plays a pivotal role in theories of fertility decline, particularly those associated with the demographic transition framework (Notestein, 1945; Freedman, 1961-62; 1963; Heer 1966; National Academy of Science 1971; United Nations, 1972, 1975). In contrast to the remarkably unambiguous empirical evidence supporting the existence of an impact of fertility on infant and child mortality, evidence of an effect of infant and child mortality on fertility has been stubbornly elusive, with different data and models yielding very different results. This occurs in spite of the fact that the potential mechanisms producing the relation are relatively clear, at least in theory (Preston, 1978). **First**, the death of an infant leads to sudden termination of

breast-feeding and this, in turn, triggers resumption of menses and ovulation thus increasing the period of exposure to a new conception. The magnitude of this purely **physiological effect** is relatively large and remarkably consistent in different demographic contexts (Jones and Palloni, 1990). Less strong but equally consistent is the effect attributed to pure cessation of lactation, that is, to termination of lactation in the absence of infant death (Delgado et al., 1982; Bongaarts and Delgado, 1977; Huffman et al., 1987; Jones and Palloni, 1990).[1] This mechanism implies a response of fertility to changes in infant mortality with lags not longer than one or two years and should be more powerful in societies where the practice of breastfeeding is pervasive and where contraception is not universally used.

The **second** mechanism, better known as the **replacement effect**, refers to couples' deliberate attempts to 'replace' any child who dies at an early age in order to attain a desired number of surviving offsprings at the end of their reproductive life. This replacement strategy serves to raise subsequent fertility and should be most evident in a context of controlled fertility (Ben-Porath, 1978; Heer and Wu, 1978; Knodel, 1978; Vallin and Lery, 1978). The mechanism, however, is believed to be less powerful than the one working through the physiological effect of breast-feeding. The empirical implication of this mechanism will again be a lagged fertility response to changes in mortality. However, the lag does not have to be necessarily as short as when the response is a purely physiological one. Indeed, evidence for replacement may become evident only at the end of the reproductive period. However, the most likely situation will be one where couples' replacement

[1] There is no contradiction between these two sets of findings. If indeed it is cessation of breast-feeding that explains resumption of menses and ovulation, it follows that the effects of breast-feeding cessation and of death of a child that truncates breast-feeding should have roughly the same magnitude. That data suggest that the latter are always larger than the former is not a threat to the theory. In fact, as argued elsewhere, the patterns of empirical results are probably due to the fact that child deaths truly truncate breast-feeding whereas cessation of breast-feeding among living children may be a flexible process, where partial continuation blends with absolute termination (Jones and Palloni, 1990).

strategy and reaction to a child's death is swift and their attempts to accelerate a conception will follow shortly after the death of a wanted child. In this case the volitional replacement response will produce effects mimicking those associated with the physiological mechanism. Indeed, a number of strategies can be employed to attempt replacement all of which leave an imprint in the length of a birth interval. Among them are earlier resumption of sexual intercourse, and cessation of contraception use. In societies practicing contraception, for example, evidence for replacement strategy will be reflected in delays in adoption or discontinuation of contraception.

Finally, a **third** mechanism may be implicated. This is the so-called **insurance (hoarding) effect** and refers to the practice of bearing more children than a desired family size even if none of the children born ever die. This protects the size of a couple's sibship against any future child death and, therefore, insures that it attains a desired family size at the end of the reproductive period. This form of anticipatory behavior can result in increases in fertility when uncertainties in the prevailing mortality environment increase or when mortality increases with certainty. Hoarding is more likely to occur in societies where children are expected to be parents' main line of old age support, to enhance reproduction of the lineage, or to reduce risk of losses to family assets, stocks or inventories. The evidence for or against the operation of this mechanism is very weak and difficult to measure with commonly available data because the insurance strategy depends on an individual couple's adjustment to **perceived** child mortality experience in society at large, and these perceptions are very hard to identify with precision. Furthermore, since hoarding is spread over the entire reproductive career of a couple and changes in perception may take a fairly long time to be translated into actual decisions, the resulting association of mortality and fertility may require a considerable time lag to

be visible and detectable.[2]

III. THE STRENGTH OF THE EVIDENCE

a. Measurement of effects and identification of mechanisms

We begin by introducing elementary indicators to draw inferences about the mechanisms linking changes in child mortality and changes in fertility from typically available empirical evidence. By and large, researchers use two related indicators of the strength of the relation. One is the elasticity of fertility relative to infant (or child) mortality and the other is a measure of replacement.

First, we define a measure of the physiological effect of an infant death. A trivial modification of an expression suggested by Preston (1978) relates either absolute or proportionate changes in infant mortality to absolute or proportionate changes in total fertility rate **solely attributable to physiological effects**.

$$\Delta TFR = ((T_2 - T_1)/T) * \Delta D \qquad (1a)$$

$$\delta TFR = ((T_2 - T_1)/T) * \delta Q * Q \qquad (1b)$$

where TFR is the total fertility rate, D is the expected total number of infant deaths among children born, Q is the infant mortality rate, ΔTFR and ΔD are absolute changes in TFR and D, δTFR and δQ are **proportionate** changes in total fertility and infant mortality respectively, T_1 is the length of a

[2] Although the terminology just introduced is fairly standard, some authors prefer to use different definitions. For example, Trussell and Olsen (1983) use the term replacement effects to include what we consider here **physiological and volitional replacement effects**. As these authors correctly point out and as we show later, empirically separating pure physiological from volitional replacement effects is a daunting task, and there is some virtue in utilizing a terminology that lumps them together. However, for conceptual clarity we will keep these two processes separate and, unless otherwise noted, we will insist on distinguishing effects that should be expected purely through physiological mechanisms and those that involve some, however primitive, conscious volitional replacement strategy.

typical birth interval following an infant death, T_2 the length of a typical birth interval in the absence of an infant death, and T is the average length of a typical birth interval. These expressions reveal two things. First, the factor $((T_2-T_1)/T)$ can be considered a measure of **physiological replacement** and is interpreted as the expected number of additional children born per additional infant death if the physiological mechanism were the only one operating. Second, the product $[(T_2-T_1)/T_1)*Q]$ measures the elasticity of fertility relative to infant mortality or the ratio of proportionate change in TFR relative to proportionate changes in Q. As the one before, this measure only reflects the physiological mechanism.[3]

T_1 takes into account **only** the reduction attributable to the shortening of the infecund period due to cessation of lactation during the first year of life of a child. Estimates of the difference (T_2-T_1) vary and are associated mainly with levels and patterns of breast-feeding. Several studies summarized by Preston suggest an **upper** bound of 13 months while evidence uncovered more recently in areas with full adherence to the practice of breast-feeding suggest values not larger than 9 or 10 (Jones and Palloni, 1990). Estimates obtained by Grummer-Strawn and Stupp from DHS data (Grummer-Strawn and Stupp, in press) are approximately 6 months for Africa, 4.5 for Asia and 3 for Latin America.[4] Because breast-feeding in Latin America has become shorter, less common and, when practiced, more erratic, it is likely that figures for the region are a lower bound, and that a more encompassing estimate would fall between 3 and 13 months, a range with a midpoint at 8 months. With a typical value of T_2 of about 24 the replacement factor amounts to about .34. With

[3] The validity of expressions (1a) and (1b) is confined to the case when changes in TFR and Q are small.

[4] These estimates were calculated as approximations from Figure 1b in the Grummer-Strawn and Stupp paper cited in the text.

levels of infant mortality around .100, characteristic during the period before the wholesale decline in mortality, we estimate that the elasticity of TFR relative to Q is of the order of .034. This means that when levels of infant mortality hover around .100, a ten percent decrease in infant mortality should induce a **physiological** response amounting to a reduction of one third of one percent (.100*.34*10) in TFR. If the value of T_2 is higher, say equal to 33, the proportionate decrease in fertility associated with a one percent decrease in infant mortality at similar levels of infant mortality is of the order of .025. For Latin American countries estimates of T_2 and T_1 of about 24 and 3 respectively are more appropriate to capture the relevant experience yielding a replacement factor close to .13 and an elasticity of .013 during periods when infant mortality was .100. The typical path followed by a Latin American country starting in 1940 involves declines in the infant mortality rate from levels near .150 to .075 during an interval of about 20 to 25 years. Assuming that the physiological effects operate alone and that the corresponding replacement factor is, as estimated before, .13, one can conclude that the decline in infant mortality accounted for not more than a 1.2 percent decrease in the TFR, from levels around 6.5 to 6.3, scarcely a significant impact during a period when TFR declines to about 5.5.

Using results from micro simulations Trussell and Olsen (1983) estimate that, under conditions that secure the absence of volitional replacement effects, the replacement factor is between .03 and .10. This range should be contrasted with the more coarse estimate suggested before, .34, and with the one we proposed as representing closely the experience of Latin American countries, .13. At levels of infant mortality of .100 the Trussell-Olsen range for the replacement factor leads to estimated elasticities of fertility relative to infant mortality not exceeding .01.

A surprising finding in this paper is that the estimates for replacement factors and elasticities

from disparate sources are fairly consistent with the idea that the effect of changes in infant mortality on TFR is very close to what one would expect from the physiological mechanism alone, that is, not much at all.

Second, we proceed to establish a relation between the elasticity of fertility relative to infant mortality and **perfect** replacement, whereby a couple is able to perfectly replace one additional child loss. That is, we attempt to infer the implied value of the elasticity of fertility in a demographic regime with perfect replacement. We then compare such implied elasticity with the value of what we called before physiologically based elasticity.

Assume that a society experiences a regime of fertility and mortality characterized by one-parameter families of fertility and mortality. Suppose that a new demographic regime is attained whereby there is one additional child death per family. If perfect replacement takes place (the replacement factor is unity) there will be one additional child born during the reproductive life of each couple. We ask, what is the magnitude of the implied elasticity of fertility if there is perfect replacement? That is, what is the ratio of the relative change in fertility required to compensate for the additional loss of a child (one extra child) to the implied relative change in infant mortality? The expression we obtain is given by:

$$\rho = ((TFR_s - D + 1)/D)^{-1} \qquad (2)$$

where TFR_s is total fertility in a suitable standard and D is the total number of child deaths experienced by a couple at the end of their reproductive period before the change in mortality (see Appendix for a full derivation). Alternative values for TFR_s and D suggest that perfect replacement

of an additional child death requires fertility elasticities between .60 and 1.20.[5] These are considerably higher than estimates of physiological effects only, and much higher than the ones we estimated for Latin American countries. But, as we shall see, these values are close to recent estimates for some areas of pre-industrial Western Europe (Galloway et al., in press).

b. Historical evidence: relations at very aggregate levels

Until the early or mid seventies, a commonly held view was that the mortality decline that started in 1950 in most developing countries would soon lead to a fertility decline. This view is consistent with the classical formulation of the demographic transition theory which, drawing from the experience of eighteenth and nineteenth century Western European countries, hypothesized a steady fertility decline concurrent with or following the process of socioeconomic development or 'modernization' (Notestein 1945). The main argument is that processes that are part of 'modernization' — industrialization, urbanization, mass education, improved health, diffusion of modern techniques, including means of birth control — are responsible for precipitating a secular decline first in adult and child mortality, and then in infant mortality, and also account for conditions favoring a secular fertility decline. The classic version of this theory does invoke but never elaborates thoroughly the direct connection between mortality and fertility change. Instead, fertility and mortality decline appear as a result of other contemporaneous processes, with the possibility that the former influences the latter mostly via a hoarding effect. A stronger version of the theory (Carlsson, 1966; Davis, 1945; Coale, 1973; Notestein, 1945) proposes that fertility reduction is, at least initially, the rational response to the realization that with lower infant and early child mortality fewer births

[5] Expression (2) is a coarse approximation since it depends on the use of (1b) but applied to changes in Q that will not normally be as small as desired to make (1b) a very accurate approximation.

are needed to secure a desired number of surviving children. Fertility reduction is an adjustment to the new mortality regime as much as an accommodation to new socioeconomic realities, such as compulsory education, which raise the direct and indirect costs of child rearing. Indeed, as suggested by Freedman (1963), a decline in mortality may be a necessary condition for substantial fertility reductions. A rather extreme version of the theory states that marital fertility reduction, and perhaps even delayed marriage, are adjustments to the pressure brought about on household (and societal) resources by reductions in early child mortality (Davis, 1963).

Analyses of data pertaining to both historical Europe and, in some cases, developing countries, cast doubts on these long-held beliefs, but particularly on the proposition that mortality decline is a precipitating cause of fertility decline. Reviewing some of the results from the Princeton fertility studies, Coale (1973) finds that reductions in mortality (whose onset was defined as the year in which infant mortality dropped by 10 percent from its maximum value) did not systematically precede the decline in fertility in many countries in Europe, as would be expected if the relations postulated by the various versions of the transition theory were indeed correct. Instead, a decline in fertility prior to or simultaneous with the drop in infant mortality was documented in all regions of France and most regions of Germany and Belgium (Coale 1973). A few years later, re-analyzing all the available data from the Princeton study Van de Walle (1986) does not find convincing statistical evidence to strengthen the proposition of a direct causal relation (Van De Walle, 1986) although, for the most part, the relations she observes are in the expected direction. Armed with more nuanced definitions of timing of fertility and mortality decline but using the same data base, Matthiessen and McCann (1978) find that child mortality did indeed begin to decline prior to fertility in almost all countries of Europe but cannot find strong evidence of an association between marital fertility and

mortality changes.

In a review of local European studies with individual (rather than aggregate) data, Knodel (1978) comes to the conclusion that the evidence is too fragile to draw any conclusions one way or the other. The only fragment of proof of indisputable value is one pertaining to the physiological effect of an infant's death. According to the estimates Knodel obtains, this effect is uniformly strong implying that "...the impact of an infant death at the onset of a birth interval shortened the average time until the next birth by 2 to 13 months" (Knodel, 1978:42). By contrast, the evidence for volitional replacement effects is simply inexistent or too weak to leave unequivocal signatures in the data analyzed.

A sobering note in the midst of these negative findings, however, comes from a very recent assessment of relations at the aggregate level. Galloway and colleagues conduct a re-analysis of relations observed in pre-industrial Europe, including countries studied in the Princeton fertility project and other data sets for Prussia, England and Wales, Sweden, and Spain that have become available in the last ten years. Their work leads to the conclusion that the bulk of estimated effects of infant mortality on fertility are, as expected, positive and that, with proper statistical procedures and when applied to data for Prussia, the relations follow expectations according to theory (Galloway et al, in press). In the various data sets they examine, Galloway and colleagues estimate the elasticity of total fertility relative to infant mortality to be within the range .50-1.55. These estimates imply that a one percent increase in the level of infant mortality leads to an increase in total fertility ranging between .50 and 1.55 percent. As shown above, these estimated elasticities **are somewhat lower than what would be required for perfect replacement** whereby the loss of a child is met by having an extra child, but higher than implied by the physiological response only. As we show later, these

estimates are also much larger than those we are able to identify for Latin American countries.

Results from other aggregate level studies discussed in a volume edited by Preston (1978) also indicate that the fertility response amounts to very incomplete replacement since "...on average, an additional child death in the family, ceteris paribus, leads to far less than one additional birth" (Preston, 1978:11). Where volitional replacement strategies could be unequivocally identified — and this occurred only in three studies pertaining to societies well into the fertility transition — the level of replacement does not exceed .50 (a child loss leads to .50 extra children). But even in these cases the estimates are suspect, in all likelihood contaminated by upward biases due to the estimation procedures used (Preston, 1978:12).

In summary, with the exception of the very recent study by Galloway and colleagues, the historical evidence from aggregate data suggests that, if anything, there is only a weak fertility response to child mortality changes, and that this is consistent with the presence of physiological-related replacement effects but not with the widespread utilization of volitional replacement or hoarding strategies.

c. Quantitative evidence from country case studies and individual level data

A number of very recent studies focus either on the experience of a single country over an extended period of time or on individual behavior in cross-sectional samples of women or couples. These studies are included in a volume prepared under the auspices of the Committee on Population, National Academy of Sciences (Montgomery and Cohen, 1997). The general inference drawn from these studies is that although there are measurable effects of infant mortality on fertility, they are seldom larger than those associated with the physiological mechanism.

Detailed studies in various countries of the developing world reveal effects of infant and child

mortality on fertility, but in most cases not as large as one would expect from physiological and replacement effects combined. Thus, for example, in a sweeping study of relations in the developing world using DHS data (Grummer-Strawn and Stupp, in press) the authors conclude that the effects of a child death is to reduce the length of a birth interval by about 30 percent — close to the replacement factor employed before and also to the one calculated by Preston (1978). However, the authors also estimate that only about 60 to 65 percent of this effect is associated with cessation of lactation and therefore attributable to the physiological mechanism only. Thus, the physiologically-related replacement rates are not larger than .195. At levels of infant mortality around .070 (the average in the countries examined in the Grummer-Strawn and Stupp paper) these figures lead to an estimated **total** elasticity of fertility relative to infant mortality of about .021, implying that the elasticity of fertility accounted for by the physiological effect alone should then be at most .014 (.195*.070).

The studies by Rosero-Bixby of the Costa Rican case (Rosero-Bixby, in press) and by Frankenberg of the Indonesian case (Frankenberg, in press) point to very weak relations between infant and child mortality and fertility. Estimates obtained by Frankenberg suggest the existence of replacement effects not exceeding those associated with the physiological mechanism. In a study of Indian data, Bhat shows that relations estimated cross sectionally indicate that only about 10 to 13 percent of the variance in total fertility rates is explained by differences in child mortality rates (Bhat, in press). At the individual level the replacement effect is estimated by Bhat to be between .02 and .10, a range even lower than in the sample examined by Grummer-Strawn and Stupp (in press) but

quite close values obtained in micro simulations under conditions of no volitional replacement.[6]

In a study of reproductive patterns in Cameroon, Kuate Defo finds a strong relation between child mortality and fertility but focuses more on the role of the death of the first two children on subsequent fertility rather that on actual estimation of overall replacement effects (Kuate Defo, in press). The evidence he gathers suggests, however, the existence of volitional replacement particularly in case of death of the first and second born. Finally, an examination of historical patterns in the US and in some regions of the US, reveals very little of significance, and the author is paradoxically forced to conclude that the relation between fertility and child mortality may have become stronger **after** the fertility transition (Haines, in press).

Despite the fact that in most of these studies the estimated effects are only slightly above or at the level expected from the physiological mechanism, there is also some evidence suggesting that the death of a child influences other components of a birth interval revealing active volitional replacement such as resumption of sexual relations (Grummer-Strawn and Stupp, in press) and contraception (Bhat, in press; Rosero-Bixby, in press; Grummer-Strawn and Stupp, in press). This is in agreement with findings obtained in Europe (Knodel, 1978) and early on in Latin America (Rutstein and Medica, 1978). Clearly, however, the implication of these findings is that, whatever these residual influences may be, they are of no more than superficial significance for otherwise they would translate into stronger replacement effects than those estimated.

IV. EVIDENCE IN LATIN AMERICA: SHORT-TERM EFFECTS DURING THE PERIOD

[6] Bhat's estimates are derived from procedures developed by Olsen (1980) and Trussell and Olsen(1983) and applied to data from two surveys of rural areas in the Southern Indian state of Karnataka. As mentioned before, Trussell and Olsen's simulation experiments suggest that the physiological effects alone should translate into 'replacement' effects amounting to between .03 and .10.

1920-1990

Suppose that replacement strategies implied a quick rather than a delayed response, perhaps spread throughout the years of the reproductive period, or that only the physiological effect operated. It should then be possible to find evidence over long periods of time of a fertility response whenever infant, and to a lesser extent child mortality experience significant changes around some secular trend. It is widely known, for example, that during periods of economic stress or duress, marriages are postponed, marital fertility is reduced, and infant and child mortality increase (Galloway, 1988; Lee, 1981; Palloni et al., 1996). Under this scenario it will generally be the case that, keeping everything else constant, periods of unusually high infant or child mortality are followed by periods of unusually high fertility. But this can occur in a number of ways that are unrelated to the impact that mortality may have on fertility. First, as marriages postponed during an economic downturn are made up in a relatively short period of time after the crisis subsides, the conditions become favorable for a surge of first births. Second, couples who postponed childbearing during the crisis resume their normal reproductive schedules and could generate a spike of some significance for births of higher order. If, however, one succeeds in controlling for these other sources of variation, the **residual** increase in fertility could be attributable to higher levels of infant or child mortality associated with the economic crisis.[7]

To test this hypotheses we propose the following model:

$$f(t)=\lambda+ \Sigma_k \alpha_k \, GDP(t-k) + \Sigma_k \beta_k \, Q(t-k) + \varepsilon(t) \qquad (3)$$

[7] The opposite responses and mechanisms are assumed to apply when instead of a crisis the society experiences an economic boom of sorts.

where f(t) is a measure of detrended births at year t, GDP(t-k) is the detrended GDP per capita evaluated at lag k, Q(t-k) is the measure of detrended infant (or child) mortality evaluated at lag k and α_k and β_k are corresponding effects. The coefficients can be interpreted as elasticities of birth relative to GDP and mortality respectively. Finally, if the error term is assumed to follow an autoregressive process of order 1, we can estimate the coefficients of the model using standard time series procedures. Even if only physiological effects predominate, we expect fertility reaction to past mortality increases to lag by three to four years. Thus, we should focus only on coefficients β associated with lags higher than three. We assume throughout that the measure of GDP per capita is sufficient as a control for influences shared by mortality and fertility.

To estimate the model we use data for the period 1920-1990 for several Latin American countries with very different demographic experiences. Argentina and Uruguay are forerunners and experience the earliest fertility and mortality decline, perhaps traceable before 1900. Costa Rica and Chile are intermediate cases with mortality declines starting not long before 1950 and a fertility decline that becomes pronounced only after 1950. Finally, Mexico is a late-comer, a country with rapid mortality decline beginning immediately during the Post World War II period but with a fertility decline that begins in earnest only in the late sixties and early seventies.

We use detrended births, detrended GDP per capita (in constant 1970 prices) and detrended infant mortality rates. We detrend the series using a local least square procedure and estimate the coefficients with a variant of the popular Cochrane-Orcutt estimator, the Prais-Weinstein estimator. Figures 1a and 1b display the series for Mexico and Costa Rica and Table 1 displays the main results. In a model with lagged infant mortality beginning at lag 3, the effects are in the expected direction and statistically significant in Costa Rica (at lag 3 but not higher), Mexico (at lag 3 but not higher)

and Uruguay (at lag 3 and marginally only at lags 4 and 5). In countries where the effects at lags three or four are of the expected sign, the elasticities are anywhere between a minimum of .023 (Chile) and a maximum of .15 (Costa Rica and Mexico). The sum of the effects over lags is a measure of the total response of fertility to mortality. It ranges from a low of .027 in Argentina to a high of about .34 in Uruguay. All estimates are below those obtained by Galloway and colleagues from cross sections in Western Europe but, according to our previous calculations, higher than expected if the mechanism is purely physiological (see above). These results are very fragile, however, since none of the sums of effects are significantly different from 0.

If, instead of using different models for each country we use a pooled data set, namely, if we impose the constrain that all effects are identical across countries, the results are slightly more robust but do not lead to different conclusions. The main results are displayed also in Table 1. According to these estimates, the elasticity of fertility relative to infant mortality is about .044 at lag 3, .015 at lag 4 and -.004 at lag 5 for an overall response of .055. Again, this exceeds what one would expect from a pure biological response (between .01 and .04 at infant mortality levels close to .100 and between .004 and .024 at levels around .075, more characteristic of the time series). This estimated elasticity implies a replacement factor close to .58, again larger than what would be expected from the physiological mechanism alone.

There are a number of problems with these estimates. First, they probably underestimate slightly the replacement effects if replacement is spread over a number of years exceeding five or six. Second, and more important, to the extent that control for other conditions affecting mortality and fertility is imperfect, and a simple measure of GDP per capita is likely to be so, the estimated effects will be biased upwards as they will also reflect fertility responses not related to mortality but to the

unmeasured conditions influencing both mortality and fertility. Third, the model assumes that undercounts and delayed registration of births and deaths roughly cancel each other out. This assumption breaks down if, for example, the magnitude of mortality surges are underestimated relative to those of fertility due to increases in undercounts directly associated with periods of economic crisis. Finally, the estimates are gross estimates of physiological and volitional replacement effects plus hoarding behavior and must be, *per force*, larger than the replacement effects associated with physiological responses alone or even physiological and volitional responses.

On the whole, these errors are likely to impart an upward bias and, therefore, to overestimate the short-term fertility response to mortality changes. Thus, the estimated total elasticity retrieved from the pooled time series (about .055) must be taken as a very generous upper boundary of the physiological and volitional responses.

V. NEW EVIDENCE: POOLED CROSS-SECTION AND TIME SERIES, 1940-1990

A different strategy of data analysis is the examination of information simultaneously across time and space. This strategy is analogous to that pursued by Galloway and colleagues and should provide comparable results. The arguments made before about the mechanisms relating infant and child mortality to fertility suggest that over time and for a particular country one should find a relation between changes in fertility during time t and changes in infant or child mortality some time before, say t-k. With the strong assumption that several countries follow an identical process, it follows that the relation should also be evident from a cross section of levels of mortality and fertility. This description of relations can be captured by the following random effects model:

$$\delta F(i,t) = \alpha + \beta\, \delta Q(i,t-k) + \gamma\, X(i,t-k) + \upsilon_I + \varepsilon_{it} \qquad (4)$$

where $\delta F(i,t)$ is the relative change in total fertility rate observed in country i during interval (t-k,t), $\delta Q(i,t-k)$ is the relative change in infant mortality rate observed in country i during the time interval (t-2k,t-k), υ_i is a fixed effect for country i, and ε_{it} is a normally distributed error term. The vector of effects γ is associated with a vector of controls X(i,t-k), and the effect β is the effect associated with infant mortality. Since fertility and mortality are measured as relative changes, the coefficients β's (and γ's) can be interpreted as elasticities and are directly comparable to estimates discussed before. For simplicity, this representation assumes that the data are arranged over equidistant periods of time and that there are no missing data.

A number of caveats are necessary. First, the model rests on the rather heavy handed assumption that there is no simultaneity, namely, that there are no contemporaneous effects of fertility on mortality, as is assumed, for example, by Galloway and colleagues. This simplifying assumption can be justified in a number of ways. The effects that fertility exerts on mortality are unlikely to be represented well by equations such as the one suggested for the effects of mortality on fertility. Indeed, it is likely that gradual changes in fertility have less of an impact than what is implied by a shift of the fertility regime. Under normal circumstances a minor or moderate decline in total fertility rate cannot not translate into immediate gains in infant or child survival. It should only do so when there are non-trivial impacts on the distribution of births by (a) length of birth intervals (previous and following); (b) parity; (c) age at birth of mothers. This is unlikely to occur in the continuous fashion suggested by a representation that includes simultaneous effects of mortality and fertility. The correct representation is probably better captured by a model where infant or child mortality is a function of a dummy variable (or set of dummies) proxying for fertility regime. But, if this is so, there is no reason to believe that simultaneity biases contaminate estimates obtained from the representation of

fertility proposed before in (4). Also, although the evidence supporting the idea that fertility exerts a strong influence on infant and child mortality is unquestionable, the actual evidence suggesting that a decline in fertility leads to a decline in mortality is elusive. This is in part due to the fact that some of the effects are offsetting (for example changes in parity distribution may increase mortality) and in part because of the simultaneous influence of related behaviors, such as lactation, which change along with fertility and mortality. A powerful example of a lack of relation can be found in the very detailed data from the registration area of Matlab (LeGrand and Phillips, 1996).

A second caveat relates to the functional transformation of the variables. As posited here, it is not the **level** of fertility that is associated with the **level** of infant (or child) mortality but their relative changes. This is akin to the preferred model used by Mathiessen and McCann (1978) but unlike most other formulations for aggregate data (Van De Walle, 1986; Galloway et al., in press). Under equilibrium conditions the difference between the two representations should be of no importance. However, once a transition gets under way and the mortality regime, for example, undergoes unequal changes in various societies, it is the model for relative changes that becomes appropriate to make inferences about the impact of infant mortality on fertility.

The third caveat pertains to the data set itself. We examine adjusted data for the period 1940-1990 in intervals of ten years thus yielding only 6 observations per country. This is a rather small number of observations to draw robust inferences about "within country effects", e.g., those that apply for a country within the observed period of time. On the positive side, however, with a few exceptions, this is the period during which all the important transformations in fertility and mortality took place.

We estimate the model on decade-specific data (adjusted for under registration) for 17

countries. The data base includes information on Total Fertility Rate, infant and child mortality (mortality rates in the first year and in the age interval 1-4), and GDP per capita as a control variable. The results of the main model appear in the first column of Table 2 ("Random Effects I"). The fit (as reflected in the value of R-Squared) is poor but the estimated effects are statistically significant and properly signed. The measure of the elasticity of fertility relative to infant mortality is .043, slightly lower than the value estimated before from the time series data for 1920-1990. The estimated effect of (the log of) lagged GDP, the control variable, is .055, positive as expected, and it too is significantly different from 0.

However, the model seem unsatisfactory on several counts. First, Figure 2 displays a graph of the relation between lagged relative changes in infant mortality and relative changes in total fertility rates. It shows that the relation between changes in infant mortality and in TFR is approximately linear when changes in the former are large enough. Second, Hausman's test reveals (highly significant value of the corresponding chi-squared statistic) that the random effects model is inappropriate since between and within effects (see column 1 in Table 2) are significantly different from each other. These two diagnostics could be related. Indeed, in a second model we introduce the idea that the size of fertility responses is related to the magnitude of changes in infant mortality. To do this we introduce an interaction term formed as the product of the lagged relative change in mortality and a dummy variable attaining the value one when infant mortality changes are larger than .010. The results are displayed in the second column of Table 2 ("Random Effects II"). The newly estimated elasticity is larger than before, about .18, when changes in infant mortality are small (interaction term equal to 0). But it is much smaller (.02) when the changes are of small magnitude (interaction term equal to 0). This is what we would expect if larger changes in mortality occur early

in the process of mortality and fertility decline, at times when use of contraception is less prevalent for in this case the response should be very close to reflecting pure physiological mechanism. The fit is roughly the same as before but, as shown by a new application of Hausman's test, the differences between coefficients from a random effects models and a fixed effects model are on the margin of being significant and warrant a rejection of the adequacy of the random effects model. To circumvent the problem we present results from a model with **within effects only** and a model with **between effects only**. The results are displayed in the final two columns of Table 2. The estimated effects associated with reveal that the bulk of the apparent fertility response to infant mortality changes is likely due to an association that exists **across countries**, not **within countries**. And, at least in a cross sectional context like the one we study, a verification of replacement effects of any kind requires that within effects be dominant. Because it is unlikely that the cross section reflects the trajectory of any one country, between effects are more likely than within effects to be due to unmeasured characteristics that affect both mortality and fertility changes and, therefore, cannot be fully trusted.[8]

In summary, this exercise with a pooled cross-section and time series suggests replacement effects in the Latin American continent cannot be too large, perhaps not larger than .18 for countries experiencing small infant mortality changes (estimate from "Random Effects Model 1") and more likely, close to .085 (estimate from "Mixed Effects (within)"). For countries experiencing large infant mortality changes the estimates are between .018 (.085-.067) and .020 (.18-.16). Again, these estimates are fragile since the estimates from the Mixed Model (within effects) are not significantly different from 0.

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[8] Alternative models with different controls (illiteracy alone; illiteracy and GDP) were also estimated but do not affect our inferences. We also use a measure of child mortality before age 5 instead of infant mortality but, here again, the results do not differ enough to warrant different conclusions.

VI. EVIDENCE IN LATIN AMERICA: INDIVIDUAL LEVEL DATA

The last strategy to estimate fertility response to changes in infant mortality relies on individual reproductive histories elicited as part of the Demographic and Health Surveys (DHS). We use two types of information. First we apply a technique suggested by Olsen (1980) and later improved by Trussell and Olsen (1983) to information on cumulated number of children ever born and surviving to mothers aged 35 and above at the time of the survey. Second, we apply hazard models and a specially designed simulation technique to data on birth intervals corresponding to events that occur during the last five years before the interview.

a. Application of the Trussell-Olsen technique

The basic elements of this technique are well-known but will be briefly reviewed here. The information required is the numbers of children ever born and dead to women who are close to or have completed their childbearing. The Ordinary Least Square (OLS) estimator of the effects of children dead on children ever born is (generally) a biased estimate of the replacement effect (volitional and physiological). Under some conditions an instrumental variable estimator (IV) is a better choice. This is calculated by first regressing the number of children dead on the proportion of children dead and then using the predicted values as regressor of children ever born. Trussell and Olsen also consider more general scenarios and diagnostic tools, including those that apply when a random coefficients model is more appropriate than either OLS or IV.

We apply their diagnostics and adjustment procedures for the DHS data of Bolivia, Brazil, Colombia, Ecuador, Guatemala, Mexico and Peru and obtain results displayed in Table 3. Even though in all cases the application of random coefficient models was questionable — their applicability was borderline — we display both the random coefficient estimates and those obtained

after correcting OLS and IV coefficients using adjustment factors suggested in Trussell-Olsen's scenario D. In addition to calculating estimates for each country we pooled together data for countries with similar breast-feeding norms. In one group we include Bolivia, Ecuador , Guatemala, and Peru, which are countries with relatively high levels of breast-feeding. In a second group we include Brazil, Colombia and Mexico, which are countries with relatively low levels of breast-feeding. As mentioned before the estimates can be interpreted as rates of replacement that reflect influences of volitional replacement as of physiological mechanisms. Note that the range of estimates in Table 3 suggests replacement factors between .02 and .40. The more robust results from the pooled samples imply replacement factors in a much narrow range, .18-.25, with a midpoint remarkably close to the estimate obtained before by setting T_1 and T_2 to 24 and 12 respectively. These replacement factors imply elasticities in the neighborhood of .022 for countries with relatively high levels of infant mortality (.100) and .015 for countries with lower levels of infant mortality (.070).

b. Estimates from information on birth intervals

To derive estimates of the relation between fertility and child mortality generally and of replacement factors in particular, we now formulate two alternative procedures that use the same information but in a somewhat different way. The first procedure follows approximately ideas first suggested by Grummer-Strawn and Stupp (in press) and requires simple manipulation of estimated effects from a hazards model. The second involves combining the results of the hazards models with a simulation yielding estimates of replacement factors (and elasticities) for a fictitious cohort that are comparable to those calculated before.

We first estimate piecewise exponential models for the risk of another conception starting with the first birth. The models take on the following form:

$$\mu_{t1\text{-}t2} = \mu_{o,t1\text{-}t2} * \exp(\beta * X_{t1}) \quad (5)$$

where $\mu_{t1\text{-}t2}$ is the hazard of a conception during the period contained between t_1 and t_2 months after the birth of the index child, $\mu_{o,t1\text{-}t2}$ is the baseline hazard, X_{t1} is a vector of covariates evaluated at t_1 , and β is a vector effects. The baseline is assumed to be exponential within the following time segments (completed months after previous birth): 1-3, 4-7, 8-11, 12-15, 16-19, 23 and over (but less than 60 by definition). We control for two background (fixed) covariates, maternal rural-urban residence and maternal education; two fecundity-related (fixed) variables, birth order and age of mother at the start of the interval; and, finally, an intermediate variable (fixed covariate) for the ever use of contraception before the start of the corresponding time segment. Our formulation departs from previous ones (Grummer-Strawn and Stupp, in press) in one respect, namely, the definition of the time dependent variables that serves to capture the death of the child that opens the birth interval. We first estimate a model with only one time-dependent covariate dummy variable (G1) that indicates whether the child who opens the interval has died before a time segment. The estimated effect of this dummy, however, confounds the physiological and volitional replacement effects. In an attempt to separate these effects we suggest a second model with a more fine-tuned categorization. We define a time dependent covariate with three categories: the first includes all those cases (birth intervals) where the child who starts the interval died before the beginning of the segment **and** its death truncates breast-feeding. A second category represents all cases where the child who opens the interval dies before the beginning of the segment but the child either never started lactation or stopped breastfeeding before death. The residual category represents all cases where the child who opens the interval is still alive at the beginning of the time segment, regardless of its breastfeeding status. We

use two dummy variables, D1 and D2, to represent, respectively, the first two classes of intervals and define the last class of births intervals as the omitted category. In theory, the effect of D1 (equal to 1 when the child who opens the interval dies and truncates breastfeeding) captures physiological influences but possibly inflated by volitional replacement effects. The effects associated with D2 (equal to 1 when the child who opens the interval dies without, however, truncating breastfeeding) can, in theory, only capture pure volitional replacement effects. We expect two regularities: a) the effects of D1 should be larger than the effects of D2, and b) the effects of a simple dummy to indicate the death of a child, G1, should be midway between the effects associated with D1 and D2.

The main results are presented in Table 4, panels a and b. Panel a displays estimated effects for the variables of interest in seven Latin American countries. These estimates were obtained with a model assuming that all effects were the same across parities. Panel b displays the effects in two pooled samples, one of countries with high levels of breastfeeding (Bolivia, Ecuador, Guatemala, Peru) and the other in countries with low levels of breastfeeding (Brazil, Colombia, Mexico). In both cases the effects of the variables representing a child's death are not constrained to be equal across parities. The following are the most important results in panel a:

i) The effects of both dummy variables are strong and (with one exception) statistically significant;

ii) The relative risks of a subsequent conception associated with a death that truncates breastfeeding (D1) range between a low of 1.79 in Ecuador to a high of 2.56 in Bolivia with a median around 2.20. Under the exponential piecewise model, increases in the baseline risk of this magnitude imply reductions in the median waiting time from birth to conception of about 8 to 9 months, close to the upper bound found in the collection if studies reviewed by Preston (1978).

iii) The relative risks of a subsequent conception associated with a death that **does not interrupt** breastfeeding (D2) are, as expected, lower than the effects of D1. They range between a low of 1.26 in Colombia to a high of 2.29 in Bolivia. These effects imply decreases in the median duration of an interval of the order of 5 months.

iv) The effects of G1 are high, statistically significant and, also as expected, midway between the effects of D1 and D2;

A plausible interpretation of these results is that the estimated effects associated with D2 primarily reflect volitional replacement, since the death of the child who opens the interval takes place after breastfeeding termination, that is, after the physiological effects have had an opportunity to act. The effects of D1 capture primarily physiological effects, but could also include the influence of replacement behaviors. To be sure, the effects of D1 are an upper bound for the magnitude of the influence of the physiological mechanism whereas those of D2 should be an upper bound for replacement effects.[9]

To calculate a range of estimated upper bounds for the physiological and volitional replacement effects we use all estimates of D1 and D2 from panel a. Under the upper bound interpretation, the estimated physiological effect translates into a reduction of the median birth interval of 8 months whereas the volitional replacement effect involves reductions of 5 months. Under this interpretation, the physiological effect is at most 62 percent (8/13) of the 'total' infant mortality effect, very close to the estimate obtained by Grummer-Strawn and Stupp (in press). The

[9] It should be clear that our definition of D1 and D2 ensures that the effects associated with D1 could be contaminated by some replacement effects whereas those of D2 could be contaminated by some physiological effects. It follows then that each of them is an upper bound for the impact of the corresponding mechanisms.

difference between the effects associated with D1 and D2 **must be a lower bound** for the pure physiological effects and 0 is a plausible lower bound for the replacement effect. With these lower bound assumptions and the information in panel awe estimate that the physiological effect amounts to reductions of the length of a birth interval not larger than 3 months and this constitutes, by assumption, 100 percent of the 'total' infant mortality effect. These lower and upper bounds yield a range of estimates for the physiologically related replacement rate between .13 and .22 when infant mortality is around .100. The **joint** effects of both the physiological mechanism **and** volitional replacement translate into reductions of birth interval of more than 3 months but less than 13 and therefore imply replacement factors in the range .13-.49 when infant mortality is around .100. Note that these ranges depend on the assumption that effects are invariant with parity and that the relation between replacement factors and birth intervals is well represented by expression (1).

Assuming that the estimated effects are accurate, what impact will a change in mortality regime have on the total level of fertility? We answer this question by implementing a second procedure that employs estimates from a hazard model in a simulation exercise. Admittedly, one could answer the question using expression (1). But this is too coarse since it assumes, among other things, that the process is the same across parities when, in fact, the effects of many variables, including mortality, are parity-specific.

To assess the effects of infant deaths on the Total Fertility Rate we adopt a procedure that consists of several steps. We **first** estimate hazard models using the same covariates as before but where their effects, including those of infant mortality **are not** constrained to be identical across parities. Since this stretches the number of events and cases too thin, we use two pooled data sets: one with countries having high levels of breast-feeding (Bolivia, Ecuador, Guatemala, Peru) and the

other with countries with low levels of breastfeeding (Brazil, Colombia, Mexico).

In a **second** step we use the estimated effects from each data set to calculate parity progression ratios (from parity 1 until parity 20) for a hypothetical women that would be observed in three different scenarios: when a child does not die, when a child dies but the death does not truncate breastfeeding and, finally, when the child dies and the death truncates breast-feeding.

The **third** step is performing a simple Monte Carlo simulation of the survival status of a child that opens an interval: a random number is drawn at each parity and an assumed level of infant mortality is used to decide if that child dies or survives during the interval. We first assume that the death of the child **always** truncates breast-feeding. This should yield an upper bound for the magnitude of the pure physiological effect. With this information and the corresponding estimated effects obtained from the hazard models we calculate the predicted parity progression ratio that would prevail if child mortality always truncates breast-feeding. We then assume that infant mortality **occurs but after breastfeeding ceases**. This should yield upper bounds for the estimates of the 'pure' volitional replacement effects. With this information and the estimates obtained from the hazard models we calculate a predicted parity progression ratio that would prevail if child mortality never truncates breast-feeding. Both calculations are repeated 20 times, from parity 1 until parity 20.[10]

Finally, the predicted parity progression ratios are chained together to estimate the (fictitious cohort) Total Fertility Rate implied by them. This is done in regimes of low, intermediate, and high

[10] In the calculation of predicted parity progression ratios all variables other than the ones associated with mortality of the child are set to their sample means. Due to non-linearities inherent in the model, this procedure will not exactly represent the mean predicted parity progression ratio. However, since we will focus on relative differences rather than on absolute values, the errors are likely to be of small magnitude.

infant mortality rates where the values of infant mortality are set equal to .035, .075 and .150 respectively. Each simulation is repeated 1,000 times producing a distribution TFR. For the purposes of our discussion, we only examine their mean values (and disregard distributional properties of the estimates).

In Table 5 we display two sets of results in the form of (mean) elasticities of TFR relative to infant mortality. One set is for countries with high levels of breast-feeding, and the other for countries with low levels of breast-feeding. Within each of these sets we have three subsets of estimated (mean) Total Fertility Rates, one for each of the three mortality scenarios, and in each one of these three subsets we have two alternative values of (mean) Total Fertility Rates depending on whether we use 'strong' effects of mortality (associated with truncation of breastfeeding) or 'weaker' effects (not associated with breastfeeding truncation). The first corresponds to expected changes when there is a transition from a regime of high mortality to one with intermediate mortality, whereas the second row corresponds to expected changes when there is a transition change from intermediate to low infant mortality. The first and third columns of numbers are elasticities corresponding to the upper bound of physiological effects whereas those in the second and fourth columns are for the upper bounds of replacement effects. The estimated elasticities associated with the physiological effect fall within a small range, .05 and .12 and, as expected, the values are higher among countries where breastfeeding is longer. The estimated elasticities associated with volitional replacement are, as expected, smaller than before and range between .03 and .10 with a mean of about .065. Under the interpretation of upper bound effects, the maximum proportionate change in TFR due to the combined influence of physiological and volitional replacement mechanisms should thus be, on average, around .14. This means that a 10 percent change in infant mortality leads to a 1.4 percent

change in TFR.[11]

VII. SUMMARY AND CONCLUSIONS

The paper reports on a number of different results based on disparate data sets. Are these results consistent or do they continue to display the ambiguity characteristic of past studies? We believe that the results of our analyses in Latin America display a fairly high level of consistency. To illustrate that this is the case we will use Table 6. This table displays alternative estimates of elasticities and replacement factors obtained from the data sources we use in the paper and compare them with alternative estimates. Since some of the sources only allow estimation of replacement factors or elasticities but not both, we transform one into the other using assumed levels of infant mortality. For clarity we identify the original estimate with the symbol '*' and, when at all feasible, we calculate ranges rather than point estimates. All results displayed in the table are valid for infant mortality levels of around .100.

First, empirical estimates of effects that reflect only the physiological mechanism as it applies in Latin America are contained within a remarkable small range, larger than .09 and smaller than .22. This range of values if higher than the values identified by Trussell and Olsen (1983) with simulated data (.02-.10) for conditions characterized by very long breast-feeding and complete absence of birth control. Neither of these two conditions is likely to describe well the bulk of Latin American countries at levels of infant mortality around .100.

Second, the estimates of the **joint** impact of physiological and volitional responses are more

[11] The definition of D1 and D2 we adopted could arguably lead to underestimates of physiological effects. This is because the residual category — that is, the cases that correspond to D1=0 and D2=0 — includes cases where the child stopped breast-feeding, even though no infant death took place. In theory, however, mothers who discontinue breast-feeding are subject to the physiological effect whether or not a child dies. To test the sensitivity of our estimates we re-estimated the hazard models with alternative definitions of the variables (results available on request). However, since they did not lead to significant changes in our conclusions, we will not report them here.

scattered and contained within a broader range, .13-.50. These values are in broad agreement with what others have found in Western Europe (Knodel, 1978; Mathiessen and McCann, 1978) and in Latin America itself (Rutstein and Medica, 1978; Rosero-Bixby, in press) but are also much lower than those found by Galloway and colleagues in Prussia (Galloway et al., in press).

The main conclusion from these estimates is that, at least in Latin America, mortality decline could not have had more than a modest impact on the fertility decline that took place after 1960 in most countries, and after 1910 among a handful of cases. The elasticities implied by Table 6 are generally of very low magnitude, particularly those identifying the physiological impact. But even if one were willing to admit the existence of some volitional replacement, a more uncertain quantity, we still would be forced to conclude that only unusually large and concentrated proportionate changes in infant mortality could have had more than trifle effects on fertility levels. This conclusion is equivalent to those drawn for Western Europe in classical studies of the Western European transition and from scattered evidence for other developing countries. It is at odds with recent analyses of evidence for Prussia. The latter analyses reveal strong effects and point to the possibility that different estimation procedures permit the analyst to identify stronger effects. But while this may be so in some parts of Western Europe, the patterns in Latin America are too consistent over time, across space, and even over units of analysis to be the result of artifacts. It is more likely that the Prussian experience is simply different, not just from other Western European countries but from the Latin American context as well.

A final issue should be highlighted. If our evidence and analyses stand scrutiny, what we have just done is to undermine the idea that fertility changes are triggered, driven, or in any way determined by changes in infant (and child) mortality. This does not mean at all that influences in the

opposite direction do not hold and, least of all, that the idea that lower mortality will be difficult to achieve without further fertility declines is inapplicable or outright invalid.

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Table 1. Estimated Short-Run Effects of Infant Mortality on Fertility.

	Effects (standard errors)			
Country	Lag 3	Lag 4	Lag 5	Sum
Argentina	-.050(.060)	.070(.060)	.007(.061)	.027 (.032)
Chile	.023(.100)	-.030(.080)	.130(.100)*	.123(.091)
Costa Rica	.146(.060)*	.009(.060)	-.016(.050)	.121(.080)
Mexico	.147(.076)*	.097(.085)	.086(.085)	.330(.232)
Uruguay	.104(.058)*	.112(.071)	.123(.104)	.339(.254)
Pooled	.044(.022)*	.015(.031)	-.004(.029)	.055(.035)

Estimates were obtained using the Prais-Weinstein estimator for autocorrelation of order 1.
* significant at $p<.01$

Table 2. Estimates from Pooled Time Series and Cross Section Models.
(Standard Errors in Parentheses)

	Model			
Estimates	Random Effects I	Random Effects II	Mixed Effects	
			Between	Within
Constant	-.24(.17)	-.17(.17)	.16(.17)	-.91(.34)
GDP	.055(.028)*	.040(.03)	-.021(.03)	.160(.06)*
Infant mortality	.043(.018)*	.180(.08)*	.36(.20)	.085(.088)
Interaction Term	-	-.160(.08)*	-.37(.20)	-.067(.10)
R-Squared (total)	.19	.20	.15	.17
Hausman chi-squared and prob. mass above it.	10.72 (.0047)	8.65 (.043)	- -	- -

Notes:

GDP is the log of the lagged value of GDP.
Infant Mortality is the lagged value of the relative change in infant mortality.
Interaction Term is the product of the dummy variable (see text) and infant mortality.
*Significant at $p<.01$, two tailed test.

Table 3. Results of Application of Trussell-Olsen Technique to Data from Women Aged 35 and Above, DHS.

Country	Preferred Estimates Adjusted OLS and IV	Estimates from Random Coefficient Models
Bolivia	.23	.58
Brazil	.47	.61
Colombia	.02	.32
Ecuador	.20	.47
Guatemala	.00	.21
Mexico	.19	.39
Peru	.20	.48
Pooled 1	.18	.50
Pooled 2	.19	.44

Table 4. Estimated Effects of Death of Child on Risk of Closing Birth Interval
(Standard Errors in Parentheses)

Panel a: Same effects across parities

	G1	D1	D2
Bolivia	.86(.06)*	.94(.11)*	.83(.07)*
Brazil	.50(.11)*	.65(.18)*	.42(.13)*
Colombia	.39(.20)	.61(.30)*	.23(.28)*
Ecuador	.53(.14)*	.58(.18)*	.30(.18)*
Guatemala	.74(.08)*	.79(.10)*	.67(.11)*
Mexico	.67(.10)*	.81(.15)*	.58(.13)*
Peru	.75(.11)*	.77(.14)*	.73(.16)*

Panel b: Different effects across parities

	Parity				
High Breastfeeding Countries	1->2	2->3	3->4	4->5	5->+
D1	.86(.13)*	.97(.13)*	.95(.13)*	.84(.15)*	.64(.09)*
D2	.85(.12)*	.92(.14)*	.51(.21)*	.90(.19)*	.45(.13)*
Low Breastfeeding Countries					
D1	1.06(.23)*	1.11(.23)*	.77(.28)*	.91(.30)*	.41(.19)*
D2	.45(.20)*	.96(.18)*	.38(.25)*	.35(.30)*	.29(.15)*

* Significant at p<.01

Table 5. Estimated Elasticities of Total Fertility Rates Relative to Infant Mortality.
(Results from Simple Montecarlo Simulations)

	Low Breastfeeding		High Breastfeeding	
	Death Dummy Variable		Death Dummy Variable	
Type of mortality change	D1	D2	D1	D2
High to Intermediate	.120	.100	.110	.090
Intermediate to Low	.064	.057	.045	.031

Table 6. Summary of Best Estimates of Replacement Factors (RP) and Elasticities (E)

	High Mortality (100 infant deaths per 1,000 births)			
	Physiological		Physiological and Replacement	
Source	E	RF	E	RF
I	.013	.13*	-	-
II	.020	.20*	.030	.30*
III	.013-.022	.13- .22*	.013-.049	.13-.49*
IV	.009-.012	.09-.12*	.020-.022	.20-22*
V	.002-.010	.02-.10*(a)	.0185	.185*(b)
VI	-	-	.050*	.50
VII	-	-	.017*	.17

I: Expressions (1) in text with T2=24 and T1=21
II: Estimates from Grummer-Strawn and Stupp for Latin American countries (DHS)
III: From estimates of effects in hazard models (no simulation and homogeneity across parities)
IV: From estimates of effects in hazard models (simulation and heterogeneity across parities)
V: (a) Trussell-Olsen estimates from their own simulated data
(b) Trussell-Olsen procedures applied to DHS data
VI: Time series data and estimated short-term effects, 1920-1990
VII: Pooled cross section and time series, 1950-1990.

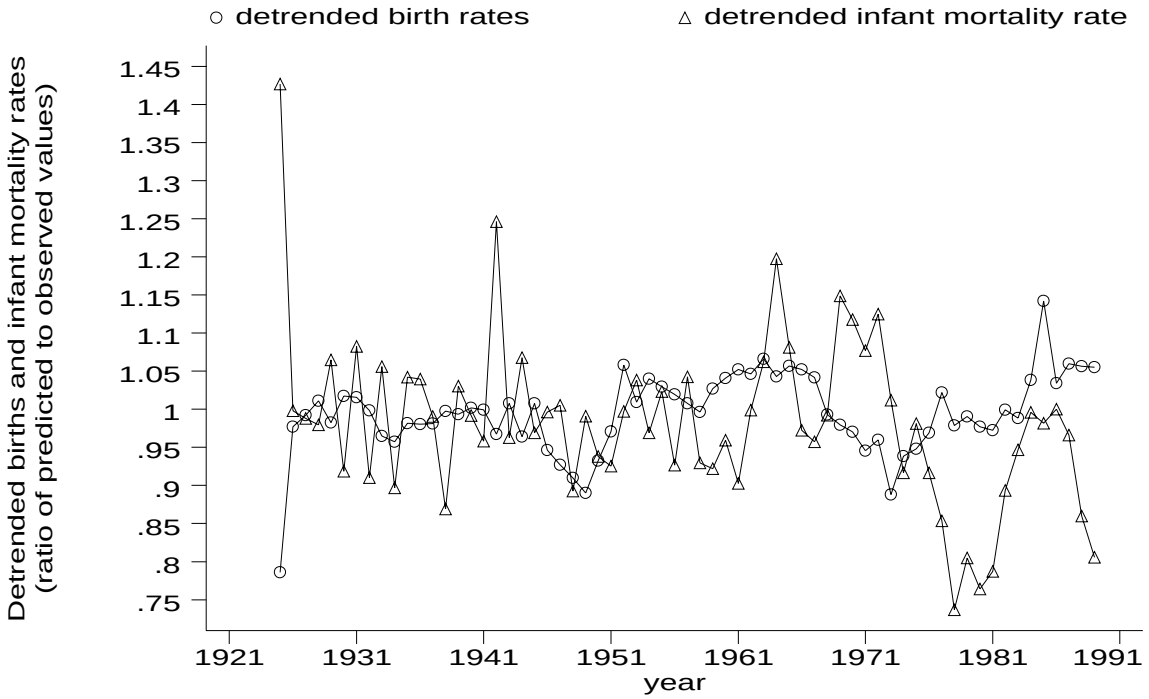

Figure 1a. Detrended Series in Costa Rica

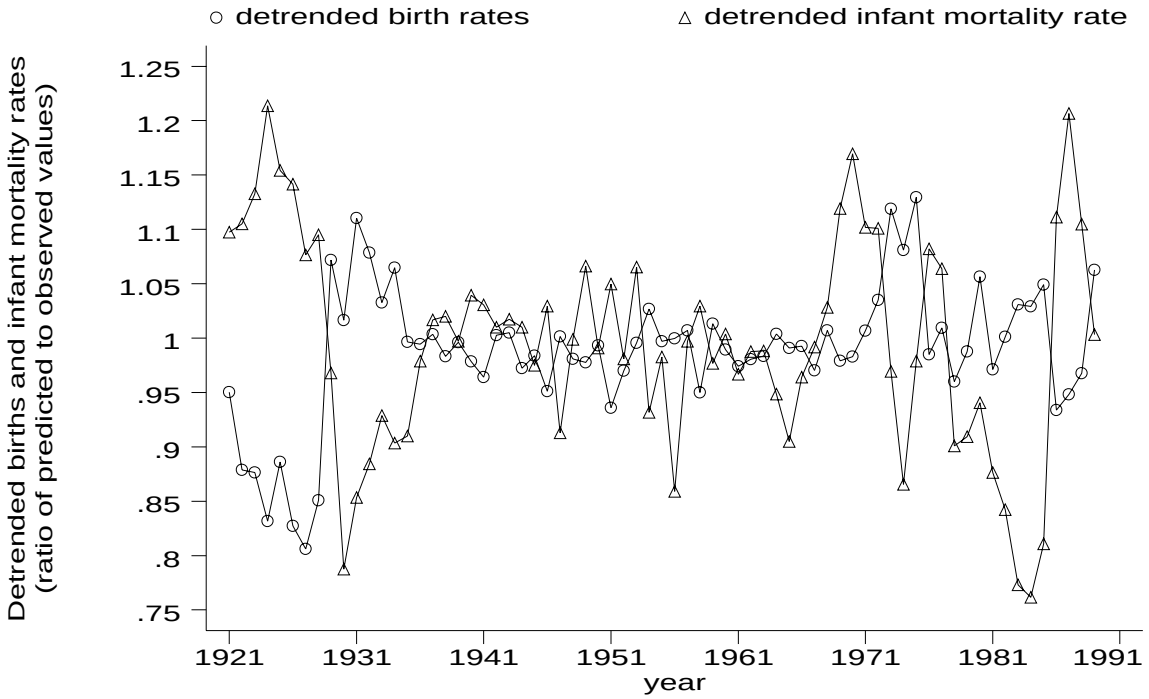

Figure 1b. Detrended Series in Mexico

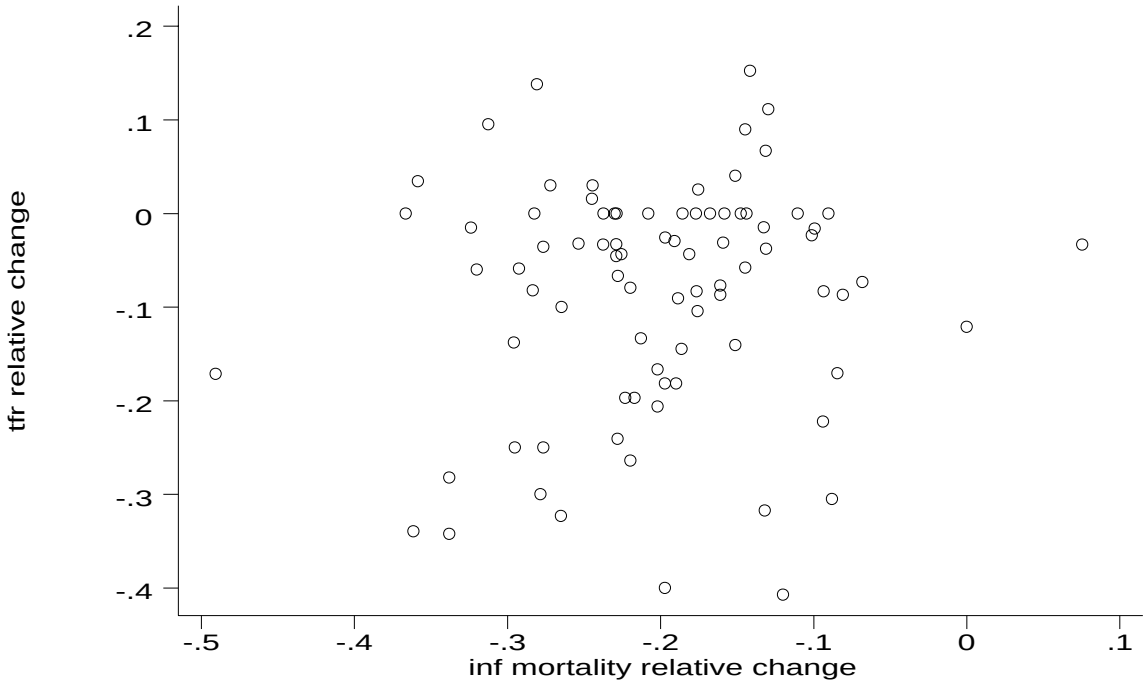

Figure 2. Fertility and infant mortality relative changes

Appendix

The problem is to derive an expression for the elasticity of the total fertility rate (TFR) relative to infant mortality, Q(0), in the case when a death of a child is replaced exactly.

1. Assume that the number of children dead among those born to women at the end of their reproductive, age t, is given by:

$$D=k*\int_x f(t-x)*Q_s(x)dx$$

where $Q_s(x)$ is the probability of dying before age x in a standard mortality schedule, k is a factor of proportionality linking actual to standard mortality and f(t-x) is the fertility rate at age t-x.

2. Suppose a shift in mortality regime increases the number of children dead by 1, so that the number of dead children is now D+1. This can be occur via change in k equivalent to $(1/D_s)$
where D_s is the number of children dead expected under the standard mortality schedule.

3. If the observed fertility is also one-parameter transformation of a standard fertility schedule, e.g., $f(y)=g*f_s(y)$, then the change in g required to compensate for an extra death is equivalent to

$$g*(TFR_s-D+1)^{-1}$$

where TFR_s is the total fertility rate associated with $f_s(x)$.

4. The original change in k implies a change in Q(0), infant mortality, equivalent to $(Q_s(0)/D_s)$ and the response of fertility to attain perfect replacement requires a change in TFR equivalent to

$$(TFR_s-D+1)^{-1}$$

This implies an elasticity equal $((TFR_s-D_s+1)/D)^{-1}$.

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