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To What Extent Does Breastfeeding Explain Birth-Interval Effects on Early Childhood Mortality?

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This article shows that in Nepal breastfeeding almost completely explains the effects of following birth interval on childhood mortality during the first 18 months of age and partially explains the effect of following birth interval on childhood mortality between 18 and 60 months of age. Breastfeeding does not explain the effect of preceding birth interval on childhood mortality. The analysis is based on an application of hazard models to data from the 1976 Nepal Fertility Survey.

In a recent article, Palloni and Millman (1986) analyzed the extent to which breastfeeding explains the effects of birth interval on early childhood mortality. Their analysis for 12 Latin American countries used logit regression and incorporated measures of both preceding birth interval and following birth interval as explanatory variables. Separate analyses were done for mortality in different age intervals. Previous birth interval was specified by two dummy variables indicating whether the previous birth interval was ≤18 months, 18–35 months, or ≥36 months. Following birth interval was specified by a single dummy variable indicating whether a following birth had been conceived by the start of the age interval. Breastfeeding was specified by a single dummy variable indicating whether the index child was breastfeeding at the start of the age interval. The investigators found that breastfeeding did not explain the effect of preceding birth interval on early childhood mortality, but that it partially explained the effect of following birth interval on early childhood mortality.

To understand why breastfeeding partially explains following birth-interval effects but not preceding birth-interval effects, it is useful to consider the particular causal mechanisms that may be operating. Regarding preceding birth-interval effects, there are at least three ways that breastfeeding of the index child might explain or appear to explain the effect of preceding birth interval on the survival of the index child. The first and most obvious way is that a short preceding interval may deplete the mother's physiological resources to breastfeed the next child. This mechanism is plausible, but we know of no evidence from previous studies that it is important. Palloni and Millman's findings suggest that it is not.

A second, less obvious mechanism involves premature births. Premature birth of the index child reduces both preceding birth interval and the survival chances of the index child. If prematurity also tends to reduce breastfeeding, then in the absence of a statistical control

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for prematurity, preceding birth interval might appear to affect survival of the index child, and breastfeeding might appear to explain some of this apparent effect. Of course, both the apparent effect and the apparent explanation of the apparent effect would be spurious. Because prematurity is rare, it seems unlikely that this mechanism could be important at the aggregate level of analysis. Palloni and Millman's findings suggest that it is not.

A third possibility is that within families, length of breastfeeding of the preceding child is positively correlated with length of breastfeeding of the index child. Because of this correlation, and because breastfeeding of the preceding child affects the duration of postpartum amenorrhea and length of preceding interval, breastfeeding of the index child might appear to explain some of the effect of preceding birth interval on survival of the index child. Again such an explanation would be spurious. It seems unlikely that this mechanism could be very important, unless the correlation between breastfeeding of the index child and breastfeeding of the preceding child is high. Again, Palloni and Millman's findings suggest that it is not important.

An explanatory role for breastfeeding seems more likely when we turn to the effect of following birth interval on child survival, because breastfeeding has direct effects on both following birth interval and child survival. Breastfeeding has direct effects on following birth interval because of its effect on postpartum amenorrhea (Bongaarts, 1983). Breastfeeding has direct effects on child survival, especially at low standards of living, because weaning may suddenly expose the child to considerably less adequate nutrition and a much higher risk of disease from contaminated water and food. Under these circumstances, it is possible that breastfeeding could explain some or all of the effects of following birth interval on child survival.

An alternative but closely related mechanism conceptualizes the following birth interval as causally prior to breastfeeding. Some women experience early pregnancy with a following child while still breastfeeding the index child, so an early following pregnancy causes early weaning of the index child instead of vice versa. In this circumstance, breastfeeding is still positively correlated with both following birth interval and child survival, and it is again possible for breastfeeding to explain the effect of following birth interval on child survival.

As mentioned earlier, Palloni and Millman did indeed find that breastfeeding partially explained the effect of following birth interval on child survival in the Latin American countries they examined. The explanatory power of breastfeeding in this context, however, was quite limited.

In this article we pursue the question of how much breastfeeding explains the birth-interval effects on early childhood mortality, using data from the 1976 Nepal Fertility Survey, which was part of the World Fertility Survey. We hypothesize that the explanatory power of breastfeeding is greater in Nepal than in Latin America, because breastfeeding is longer, mortality is higher, and contraception is rarer in Nepal. Under these circumstances breastfeeding has strong effects both on child survival and on birth interval through its effect on postpartum amenorrhea. We hypothesize, in addition, that in Nepal, as in Latin America, breastfeeding does not explain the effect of previous birth interval on early childhood mortality. We expect this because we know of no evidence that either maternal depletion or prematurity is unusually common in Nepal. Finally, we hypothesize that in Nepal breastfeeding of the index child contributes substantially to explaining the effects of following birth interval on early childhood mortality through the early weaning mechanism. We expect this because breastfeeding has a substantial effect on birth interval in Nepal and because nutrition, sanitation, and medical services are comparatively poor there. Under these conditions, early weaning typically has a substantial adverse effect on early childhood mortality.

(For further discussion of the mechanisms by which birth interval affects early childhood mortality, see DaVanzo, Butz, and Habicht, 1983; Hobcraft, McDonald, and Rutstein, 1985; Pebley and Stupp, 1986.)

Socioeconomic covariates included in the analysis are mother's literacy, father's literacy, rural–urban residence, and region (hill, terai, mountain). The NFS did not include a question on income; but in the present context this may not be a serious omission, because previous studies in other countries indicate that the effect of income on childhood mortality is often greatly reduced when mother's and father's education or literacy is taken into account (Cochrane, O'Hara, and Leslie, 1980; DaVanzo, Butz, and Habicht, 1983; Mensch, Lentzer, and Preston, 1985).

Six demographic control variables are included in the analysis. The first of these is sex, represented as a dummy variable (1 if male, 0 otherwise); because of sex differentials in mortality and the high degree of son preference in Nepal, it seemed appropriate to control for this variable. Maternal age has been shown to have effects on childhood mortality and is included to control for variability in mother's age at childbirth. Maternal age in years is treated as a continuous variable. Since the effect of maternal age on early childhood mortality tends to be curvilinear, with mortality higher at young and old maternal ages and lower in between, maternal age squared is also included.[1] Birth order tends to be correlated with childhood mortality and therefore appears as an additional demographic control in the models. Birth order squared is also included, to allow for the possibility that the effect of birth order on early childhood mortality increases at very high birth orders. Another demographic covariate is whether the preceding child survived to the time of the survey. Without a control for survivorship of the preceding child, the measured effect of preceding birth interval is biased, because survivorship of the preceding child may affect postpartum amenorrhea and preceding birth interval. Survivorship of the preceding child also serves as a partial control for previous child mortality within the family.

The principal independent variables included in the models are the birth-interval and breastfeeding covariates. The birth-interval covariates are subdivided into two categories, the first pertaining to preceding birth interval and the second pertaining to following birth interval. Preceding birth interval is treated as a continuous variable. Because birth-interval effects on childhood mortality tend to be curvilinear, such that a 1-month increase in birth interval tends to have a progressively smaller effect on mortality as the birth interval lengthens (Hobcraft, McDonald, and Rutstein, 1985), preceding birth interval squared is also included.

Following birth interval is specified somewhat differently, as following birth status in the form of two dummy variables—"following birth surviving" and "following birth dead." The reference category is "no following birth as yet." The two dummies are specified as age-varying covariates in the hazard models and are always defined one month prior to the current life-table age of the index child. In effect, for mortality in each age interval, we look one month before and ask, Has the next birth arrived yet and, if so, is it alive or dead? Then what is the effect of this following birth status on the mortality risk for the index child in the next age interval? Although the values of the dummies may vary by age for a particular child, the model is specified so that the coefficients of the dummies are invariant by age. Thus the coefficients represent average effects of following birth status over all ages included in the life table (0–18 or 18–60 months, depending on which dependent variable is being considered). The specification of effects as averages over all ages has the advantage of reducing the sensitivity of the results to bias from age misreporting.

Breastfeeding is also included as an age-varying covariate, and it is also coded as a dummy variable (1 if breastfeeding, 0 otherwise). In the hazard models, at each age of child, the risk of death depends on breastfeeding status one month earlier. Thus just as in the case of the following birth status covariates, the value of the breastfeeding covariate may change for a given child as the child gets older, and the coefficient of breastfeeding represents the average effect of breastfeeding over all ages included in the life table.

Breastfeeding one month prior is meaningless when the index child is less than 1 month old. In this case, breastfeeding is coded "yes." Since there are no doubt a few children who

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died in the first month for whom a "no" code would have been appropriate, the uniform assumption of "yes" introduces a small conservative bias in the estimated effect of breastfeeding (Choe et al., 1989). Undoubtedly a sizable fraction of the "no" cases, however, were never breastfed only because they were too sickly at birth and died soon thereafter. In such instances, a "yes" code is not unreasonable, because the lack of breastfeeding is appropriately viewed as caused by infant mortality rather than the other way around.

The covariates are employed in a series of hazard models. The computer program is BMDP2L. Partial likelihood is used for estimation. It has been shown that partial likelihood can be treated like the usual maximum likelihood (Tuma and Hannan, 1984:244–247).

Results

Table 1 shows the mean values of the covariates employed in the models. In the case of dummy variables, the means are expressed as percentages. The table shows that the sample is heavily skewed toward illiterate mothers, rural residence, and non-mountain residence. This means that the variables for mother's illiteracy, rural residence, and mountain residence cannot explain much of the variability of child mortality in the sample, even if the effects of these variables on child mortality are substantial. This problem is not serious for the other covariates.

Infant Mortality (0–18 months)

Table 2 presents seven hazard models of infant mortality. In the models, year of birth, the block of socioeconomic covariates, and the block of demographic covariates are treated

Table 1. Socioeconomic and Demographic Characteristics of Births, 1975 Nepal Fertility Survey

Characteristic	Percentage	Average
Mother illiterate	94.7	
Father illiterate	56.1	
Rural residence	97.5	
Region		
Hill	51.1	
Terai	41.4	
Mountain	7.5	
Sex (male)	51.0	
Maternal age (years)		28.9
Birth order		4.4
Preceding child dead	25.8	
Preceding birth interval (months)		35.8
Breastfed at least 15 months	78.0	

Note The average age of preceding children at the time of the survey was 61.8 months. The percentage breastfed at least 15 months was calculated by the life-table method. In computing the life table, infant deaths and following births as well as the event of reaching the survey date were treated as censoring events.

Table 2. Effects of Socioeconomic and Demographic Characteristics on Infant Mortality (0–18 months): Hazard Model Estimates of Relative Risk

Characteristic	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Year of birth	0.9772	0.9804	0.9785	0.9736*	0.9783	0.9737*	0.9757*
Socioeconomic covariates							
Mother illiterate	1.3754*	1.3513	1.4032*	1.4175*	1.4028*	1.4176*	1.3753*
Father illiterate	1.0232	1.0137	1.0296	1.0378	1.0290	1.0382	1.0221
Rural residence	1.0463	1.0465	1.0625	1.0667	1.0619	1.0670	1.0449
Region							
Hill	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
Terai	1.0088	0.9981	0.9751	0.9834	0.9753	0.9834	1.0063
Mountain	1.2602*	1.2506*	1.2277	1.2427*	1.2290	1.2417*	1.2680*
Demographic covariates							
Sex (male)	0.9863	0.9912	1.0024	0.9991	1.0023	0.9992	0.9867
Maternal age (years)	0.9282	0.8791***	0.8789***	0.9269	0.8789***	0.9270	0.9281
Maternal age squared	1.0010	1.0016**	1.0016**	1.0011	1.0016**	1.0011	1.0010
Birth order	1.0220	1.1212	1.1249	1.0252	1.1248	1.0252	1.0213
Birth order squared	1.0040	1.0006	0.9997	1.0030	0.9997	1.0030	1.0039
Previous child dead	1.4740****	1.5804****	1.5517****	1.4552****	1.5526****	1.4545****	1.4754****
Birth interval and breastfeeding							
Preceding birth interval (months)							
Preceding birth interval	0.9764****	—	—	0.9785****	—	0.9785****	0.9767****
Preceding birth interval squared	1.0001	—	—	1.0001	—	1.0001	1.0001
Following birth							
No following birth as yet	—	1.0000	—	1.0000	1.0000	—	1.0000
Following birth surviving	—	3.7083****	—	1.1043	1.1208	—	3.5194****
Following birth dead	—	3.3897	—	0.9442	0.9947	—	3.0916
Breastfeeding	—	—	0.1857****	0.1947****	0.1880****	0.1927****	—
Likelihood statistics							
−2(log-likelihood)	13,867.1	13,910.0	13,806.3	13,754.8	13,806.2	13,754.9	13,854.8
Difference from model 4	112.2 (3)****	155.2 (3)****	51.5 (4)****	—	51.4 (2)****	0.1 (2)	100.0 (1)****
df	14	14	13	17	15	15	16

Notes: Significance tests are two-tailed. Numbers in parentheses are differences in degrees of freedom from model 4.
* Significant at the 10 percent level.
** Significant at the 5 percent level.
*** Significant at the 1 percent level.
**** Significant at the 0.1 percent level.

as controls. The effects of these variables are generally consistent with findings from earlier studies and are not discussed in detail here. The block of birth-interval and breastfeeding covariates contains the independent variables of principal interest. As already mentioned, this block is subdivided into three subcategories corresponding to preceding birth interval, following birth status, and breastfeeding. In models 1–3, the effects of these three subcategories on infant mortality are examined one at a time. Model 4 is the full model. Relative to model 4, model 5 deletes the preceding birth-interval covariates, model 6 deletes the following birth-status covariates, and model 7 deletes the breastfeeding covariates. Comparison of the seven models yields assessments of how the effects of birth interval and breastfeeding on infant mortality are affected by each block of covariates and by all covariates simultaneously.

Before proceeding to the actual findings, a few additional comments on the format of Table 2 are in order. The effects of the various covariates are expressed as relative risks. For example, in the full model (model 4), the relative risk of 0.9736 for year of birth means that the effect of a 1-year increase in year of birth is to multiply the hazard function (i.e., the age-specific mortality risks) by 0.9736. The relative risk of 1.4175 for mother illiterate means that age-specific mortality risks for illiterate mothers are 1.4175 times higher than age-specific mortality risks for the reference category of literate mothers.

The effects, or relative risks, are calculated as $\exp(b_i)$, where b_i is the underlying estimated coefficient for the ith covariate. (The underlying coefficients are not shown in the tables.) The symbols indicating level of significance refer to the departure of the underlying coefficients from zero or, equivalently, the departure of the relative risks from one. The observed levels of significance shown in the tables are almost certainly low, considering the number of cases, because the data suffer from large variances resulting from considerable statistical noise generated by age misreporting. Thus it could be argued that the estimated effects, interpreted as average effects over an 18-month age interval, are more statistically significant than they appear to be at first sight from the standard tests. (Of course, there could also be an offsetting bias in the opposite direction due to the application of methodology for simple random sampling to a cluster sample.)

Proceeding now to the findings in Table 2, we see from model 1 that preceding birth interval has a moderately large, highly statistically significant effect on infant mortality. For example, the effect of a 10-month increase in preceding birth interval, from 30 to 40 months, is to multiply age-specific mortality risks by $[(0.9764)^{10}][(1.0001)^{700}] = 0.8447$, amounting to a 15 percent reduction (the exponent of 700 is calculated as $40^2 - 30^2$).[2] Model 2 shows that following birth has a much larger effect on age-specific mortality risks. The effect is highly statistically significant if the following birth was still alive 1 month before. The effect of a following birth, relative to no following birth as yet, is to multiply age-specific mortality risks by 3.4 if the following birth was dead 1 month previously and by 3.7 if it was still alive 1 month previously. The lack of statistical significance for the relative risk of 3.4 for following birth dead is probably due mainly to the much smaller number of cases for which the following birth was dead than those for which the following birth was surviving. Model 3 shows that breastfeeding has a huge effect on infant mortality. The effect of breastfeeding, relative to not breastfeeding, is to multiply age-specific mortality risks by 0.1857, representing a mortality reduction of almost 82 percent. The effect is highly statistically significant.

In model 4, all three subcategories—preceding birth interval, following birth status, and breastfeeding—are entered into the model simultaneously. The effects of preceding birth interval and breastfeeding are virtually unchanged. This is consistent with Palloni and Millman's earlier finding that breastfeeding does not explain the effect of preceding birth interval on infant mortality. The effects of the following birth covariates, however, are drastically reduced, to values close to 1 that are statistically nonsignificant. Thus it appears

that the effects of the following birth covariates on infant mortality are explained almost completely by breastfeeding, in contrast to Palloni and Millman's findings for Latin America.

This interpretation is strengthened by findings from models 5–7. Model 5 deletes only the block of preceding birth-interval covariates, relative to model 4, the full model. When the preceding birth-interval covariates are deleted, the effects of the following birth covariates and breastfeeding hardly change. Model 6 deletes only the following birth-interval covariates; when this is done, the effects of preceding birth interval and breastfeeding hardly change. Model 7 deletes breastfeeding. Again the effect of preceding birth interval hardly changes, but now the effect of following birth interval increases dramatically. Models 5–7 show that it is breastfeeding and not preceding birth interval that explains away the effects of the following birth covariates in model 4. The likelihood statistics show, in addition, that all models except model 6, which deletes only the following birth covariates, differ significantly from the full model. This finding also suggests that breastfeeding accounts almost completely for the effects of the following birth covariates.

The obvious interpretation of the finding that the effect of a following birth on infant mortality is felt almost completely through breastfeeding is that a short following birth interval is associated with early weaning, for reasons discussed earlier, and that early weaning greatly increases the risk of getting sick and dying. This is not surprising in Nepal, where nutrition is often worse after weaning than before, water and food are frequently contaminated, and sanitation and medical services are generally poor (Barber, 1986; Blaikie, Cameron, and Seddon, 1979, Economic and Social Commission for Asia and the Pacific, 1984; Nepal Central Bureau of Statistics, 1984, World Bank, 1983, 1987).

Child Mortality (18–60 Months)

We turn now to the models of child mortality in Table 3. As expected, the determinants of child mortality differ in relative importance from the determinants of infant mortality in Table 2.

Table 3 is identical in format to Table 2. Again the strategy in models 1–3 is to introduce the three blocks of birth-interval and breastfeeding covariates one block at a time. Model 1 shows that preceding birth interval no longer has an effect on child mortality. Model 2 shows that following birth has a substantial effect on mortality, but much more so if the following child has died than if it has survived. If the following child has died, the hazard is more than three times higher than the reference category (no following birth as yet), and the effect is highly statistically significant. If the following child has survived, the hazard is 30 percent higher, but the effect is not statistically significant. Model 3 shows that breastfeeding again has a major effect on mortality. Relative to not breastfeeding, breastfeeding reduces mortality by about 55 percent. As expected, the effect of breastfeeding is much smaller than in the case of infant mortality.

Model 4 is the full model. Again preceding birth interval has no effect, and the effect of a following birth on mortality is greatly reduced. The relative risk for following birth surviving is reduced from 1.30 to 0.91, neither of which is statistically significant, and the relative risk for following birth dead is reduced from 3.08 to 2.23; the value of 3.08 is highly significant, and the value of 2.23 remains significant at the 5 percent level. The relative risk for breastfeeding is virtually unchanged. The results show that breastfeeding helps explain the effect of following birth dead, but the explanation is not complete. This conclusion is strengthened by the results from models 5–7 and the likelihood statistics.

Model 4 for child mortality (Table 3) may be compared with model 4 for infant mortality (Table 2) with regard to the effects of following birth status. In Table 2, there is little difference between the effect of following birth surviving and the effect of following birth dead on infant mortality. Neither effect is statistically significant. But in Table 3, the effect of following

Table 3. Effects of Socioeconomic and Demographic Characteristics on Child Mortality (18–60 months): Hazard Model Estimates of Relative Risk

Characteristic	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Year of birth	0.9533	0.9517	0.9463	0.9470	0.9491	0.9441	0.9486
Socioeconomic covariates							
Mother illiterate	1.8273	1.7750	1.8833	1.8416	1.8358	1.8886	1.7863
Father illiterate	1.3702**	1.3555*	1.3639**	1.3569*	1.3538*	1.3669**	1.3588*
Rural residence	4.2357	4.1851	4.4558	4.5200	4.4771	4.4944	4.2426
Region							
Hill	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
Terai	0.7127**	0.7115**	0.6649***	0.6700**	0.6690***	0.6665***	0.7125**
Mountain	0.7512	0.7804	0.7493	0.7604	0.7634	0.7467	0.7769
Demographic covariates							
Sex (male)	1.0921	1.0896	1.0921	1.0876	1.0871	1.0926	1.0895
Maternal age (years)	1.0201	1.0025	1.0017	1.0190	1.0053	1.0149	1.0222
Maternal age squared	0.9994	0.9996	0.9997	0.9995	0.9996	0.9996	0.9994
Birth order	1.1481	1.1818	1.1565	1.1283	1.1554	1.1298	1.1431
Birth order squared	0.9923	0.9909	0.9927	0.9930	0.9923	0.9935	0.9920
Previous child dead	1.4970***	1.4878***	1.5362***	1.4878***	1.5039***	1.5210***	1.4634**
Birth interval and breastfeeding							
Preceding birth interval (months)							
Preceding birth interval	0.9913	—	—	0.9946	—	0.9950	0.9920
Preceding birth interval squared	1.0000	—	—	1.0000	—	1.0000	1.0000
Following birth							
No following birth as yet	—	1.0000	—	1.0000	1.0000		1.0000
Following birth surviving	—	1.3022	—	0.9098	0.9159	—	1.2781
Following birth dead	—	3.0826****	—	2.2293**	2.2315**	—	3.0515****
Breastfeeding	—	—	0.4468****	0.4562****	0.4506****	0.4531****	—
Likelihood statistics							
−2(log-likelihood)	3,073.9	3,065.7	3,051.3	3,044.7	3,045.5	3,050.5	3,064.2
Difference from model 4	29.2 (3)****	21.0 (3)****	6.6 (4)	—	0.8 (2)	5.8 (2)*	19.5 (1)****
df	14	14	13	17	15	15	16

Note: Significance tests are two-tailed. Numbers in parentheses are differences in degrees of freedom from model 4.
* Significant at the 10 percent level.
** Significant at the 5 percent level.
*** Significant at the 1 percent level.
**** Significant at the 0.1 percent level.

birth dead on child mortality is significant at a relative risk of about 2.2. The effect of following birth surviving is slightly below unity and is not significant. These findings suggest that in the case of infant mortality, the removal of competition for maternal attention (when the following child dies) tends to lower mortality risks for the index child, thereby offsetting the effects of infection that may be associated with the death of the following child. In the case of child mortality, on the other hand, the results suggest that the index child is old enough and independent enough that the removal of competition for maternal attention has little effect on mortality risks, thereby not offsetting the effects of infection associated with the death of the following child, and therefore leading to a significant positive effect of following birth dead Another possibility relates to the fact that in the case of infant mortality, but not child mortality, the following children who die are uniformly very young (0–3 months of age, since the following birth interval can range only from 9 to 12 months), so a substantial fraction of these deaths are from causes other than infectious disease. Therefore, the infectious disease effect of following birth dead may be considerably larger for child mortality than for infant mortality.

The finding in Tables 2 and 3 that father's literacy has no effect on infant mortality but a substantial effect on child mortality is also worthy of comment. This finding suggests that as long as the child is on the breast, it receives assured nourishment independently of father's socioeconomic status as reflected by father's literacy. When the child is weaned, however, its health then also depends on nourishment apart from breastmilk, and the availability and quality of such nourishment is influenced by the socioeconomic status of the family as reflected by father's literacy. In Nepal, children aged 18–60 months are much more likely to be weaned than children aged 0–18 months. Thus this line of reasoning leads to the expectation, confirmed in the Nepal findings, that father's literacy affects child mortality much more than it affects infant mortality.

Conclusion

In Nepal, both preceding birth interval and following birth interval have substantial effects on infant mortality (defined here as mortality at the ages of 0–18 months), before the introduction of breastfeeding as an explanatory variable. Breastfeeding explains virtually none of the effect of preceding birth interval but almost all of the effect of following birth interval. Our interpretation of the latter finding is that a short following birth interval tends to be associated with early weaning of the index child, and that conditions in Nepal are such that early weaning has deleterious effects on health, stemming mainly from poorer nutrition following weaning and from disease contracted from contaminated water and food.

Birth-interval effects are smaller in the case of child mortality, defined here as mortality at the ages of 18–60 months. Preceding birth interval no longer affects child mortality. Following birth interval still has substantial effects, but only if the following child has died. Breastfeeding only partially explains the effect of following birth dead.

The findings thus indicate that at the ages of 18–60 months, breastfeeding is not the only avenue through which following birth interval affects child mortality. Another likely avenue involves the possibility that the index child will catch and die from the same disease that the following child died from. It is puzzling, however, that this mechanism is not evident in the case of infant mortality. Perhaps this discrepancy occurs because, in the case of infant mortality, removal of the following child through death reduces competition for maternal attention for the index child, thereby lowering mortality risks for the index child. This offsetting effect may be largely absent for children older than 18 months, who are less dependent on maternal attention. Another possibility is that the infectious disease effect on the index child, stemming from the death of a following child, is smaller in the case of infant mortality than in the case of child mortality. This possibility exists because, in the

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case of infant mortality, the following children who die are extremely young, when death often occurs from causes other than infectious disease.

Our findings differ to some extent from those of Palloni and Millman, who found for 12 Latin American countries that breastfeeding did little to explain following birth-interval effects on early childhood mortality. The greater explanatory power of breastfeeding in Nepal probably occurs because breastfeeding is longer, mortality is higher, and contraception is rarer. Consequently, breastfeeding has strong effects on both child survival and birth interval through its effect on postpartum amenorrhea.

It is also possible, but less likely in our judgment, that differences in model specification account for the differences in findings. Our hazard models use a more elaborate specification of following birth and breastfeeding effects than Palloni and Millman used in their models. Moreover, the time sequence between cause and effect, specified by 1-month lags in our hazard models, is tighter. Palloni and Millman specified following birth interval as a single dummy variable indicating whether a following birth has been conceived at the start of the age interval, regardless of the length of the age interval. Breastfeeding was likewise specified as breastfeeding status at the start of the age interval, regardless of the length of the age interval.

A noteworthy by-product of the analysis is the finding that breastfeeding effects on early childhood mortality are very large in Nepal. Relative to not breastfeeding, breastfeeding reduces infant mortality by about 80 percent and child mortality by about 55 percent in the full models. These very large effects must be interpreted cautiously, because they may be exaggerated to some extent by simultaneity bias. Simultaneity bias can occur if dying children stop breastfeeding because of illness one month or more before death, as pointed out previously by Habicht, DaVanzo, and Butz (1986). In such instances, the cessation of breastfeeding is more appropriately viewed as a consequence than a cause of mortality. Further research is needed to assess the magnitude of this possible bias.

Notes

[1] Without maternal age squared, the model would be

$$h(x, A, Z) = h_0(x)\exp(bA + \mathbf{d}Z),$$

where $h(x, A, Z)$ is the hazard, $h_0(x)$ is the baseline hazard, A is maternal age, Z is a column vector of the remaining independent variables, and d is a row vector of coefficients of the remaining independent variables.

In this simple model, the multiplicative effect on the hazard of a one-unit increase in A is

$$\frac{h(x, A + 1, Z)}{h(x, A, Z)} = \frac{h_0(x)\exp[b(A + 1) + \mathbf{d}Z]}{h_0(x)\exp[bA + \mathbf{d}Z]} = \exp(b)$$

The multiplicative effect of a one-unit increase in A on the hazard is independent of A.

The simplest way to introduce curvature (dependence on A) is to introduce a quadratic term, just as in ordinary multiple regression

$$h(x, A, Z) = h_0(x)\exp(bA + cA^2 + \mathbf{d}Z)$$

The multiplicative effect of a one-unit increase in A on the hazard is then

$$\frac{h(x, A + 1, Z)}{h(x, A, Z)} = \frac{h_0(x)\exp[b(A + 1) + c(A + 1)^2 + \mathbf{d}Z]}{h_0(x)\exp[bA + cA^2 + \mathbf{d}Z]}$$

$$= \exp[b + c(2A + 1)].$$

The multiplicative effect of a one-unit increase in A on the hazard now depends on A. When c is positive, the graph of $h(x, A + 1, Z)/h(x, A, Z)$ against A is concave upward and increasing; when c is negative, the graph is concave downward and decreasing. A quadratic term is appropriate when

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one's theory predicts a simple curvature that is one of these two kinds. The hypothesized curvature need not be symmetric for a quadratic term to be appropriate.

[1] The model can be represented as

$$h(x, I, \mathbf{Z}) = h_0(x)\exp(bI + cI^2 + \mathbf{dZ}),$$

where I denotes the preceding birth interval. Then

$$\frac{h(x, 40, \mathbf{Z})}{h(x, 30, \mathbf{Z})} = \frac{h_0(x)\exp[40b + (40^2)c + \mathbf{dZ}]}{h_0(x)\exp[30b + (30^2)c + \mathbf{dZ}]}$$

$$= [\exp(b)]^{10}[\exp(c)]^{(40^2 - 30^2)}.$$

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