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Comment on Pampel and Pillai's "Patterns and Determinants of Infant Mortality in Developed Nations, 1950–1975"

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Pampel and Pillai (1986) examined a number of potential determinants of infant mortality rates (IMRs) in a pooled cross-section time series sample of developed nations over the period 1950–1975. In multivariate analyses, they regressed infant mortality rates on the percentage of live births born to mothers below 20 years of age, along with controls for economic, social welfare, and medical care aspects of the countries and time periods considered. In their conclusion, they offered several policy recommendations to help reduce infant mortality rates in the United States:

> It is an oversimplification to blame the problem [infant mortality] on the medical care system, federal programs, and inequitable income distribution; they are not the only or most important sources of high rates in the U.S. . . . Our results therefore provide a basis on which to make policy changes to reduce the IMR. Little can be done to change ethnic diversity, but economic growth, postponement of fertility, and access to hospital beds may be useful. In addition, improved female education, although already high in the U.S. compared with other nations, would bring large returns, reducing infant mortality directly through healthier life styles and indirectly through lower teenage fertility. (p. 537)

We question both the belief that postponed fertility (lower teenage fertility) will reduce infant mortality in the United States and the omission of an increase in social welfare expenditures from these policy recommendations. These questions arise from, first, an alternative interpretation of the information that their "teen births" variable actually reflects; second, direct evidence regarding the likely contribution of teen births to neonatal mortality in the U.S.; and finally, a straightforward reading of Pampel and Pillai's empirical results.

Implicit in promoting a policy of delayed fertility is the assumption that the positive (partial) association between early fertility and infant mortality is in some sense causal—that is, that the characteristics associated with teen motherhood that lead to excessive infant mortality could be avoided or "outgrown" if those women who actually bear children in their teens were to postpone childbearing until their 20s.

Here we offer an alternative interpretation of this association that implies that infant mortality rates may not be improved by a policy of delayed fertility. Given that both teen birth rates and IMRs are known to be high among poverty populations, and since Pampel and Pillai do not directly control for poverty births in their analysis,[1] it is possible that the effects of poverty are loading onto the teen births variable. We hesitate to interpret the coefficient on the teen births variable as a partial effect of teen births on the IMR *independent of poverty status*, since teen births may be Pampel and Pillai's best measure of population segments characterized by both early fertility and poverty.

Consistent with our view that the positive association between teen births and infant mortality may be due to unmeasured poverty status, Pampel and Pillai noted a "strong correlation between [per capita] GNP and percent teenage births ($r = 0.645$) and a weak negative relationship between teenage births and the TFR ($r = -0.102$). As national income grows and fertility declines, a growing proportion of births occur to teenagers" (Pampel and Pillai, 1986:538). But why should the percentage of births to teens increase as per capita gross national product (GNP) increases unless those populations (within countries) who may not enjoy the benefits of increases in national income, the persistently poor, continue to have children at young ages while more advantaged subpopulations experience fertility declines and delayed childbearing? In fact, although there is evidence of a general trend in the U.S. toward delayed childbearing (e.g., Bloom and Trussell, 1984), there is also evidence that the disadvantaged in the U.S., particularly poor black Americans, initiate and complete their fertility at young ages (Brooks-Gunn, Furstenberg, and Morgan, 1986; Evans, 1986; Millman and Hendershot, 1980). In short, as GNP increases and fertility declines, the composition of the pool of childbearers changes such that a larger percentage of births are to poor mothers who tend to bear children in their teenage years.

Therefore, when considering a policy of postponement of fertility as a means of reducing infant mortality, we should be cautious in concluding that in the counterfactual world in which all women postpone childbearing, poor women would enjoy improved infant outcomes. There is a growing body of research challenging the notion that teenage motherhood is detrimental to infant survival, independent of socioeconomic status (Geronimus 1986a; for reviews, see Geronimus, 1987; Makinson, 1985). In fact, there is evidence that within populations for whom early fertility is characteristic, postponement of childbearing into the 20s may *increase* neonatal mortality rates (Geronimus, 1986a), perhaps because longer exposure to poverty increases the reproductive risk status of mothers, or because some of the physiological effects of early exposure to poverty can be irremediable or progressive, even for those who manage to escape poverty (Geronimus, 1986b, 1987).

As an empirical assessment of the potential effect of a policy of delayed childbearing on the U.S. infant mortality rate, Table 1 presents neonatal mortality rates (NMRs) by age and race of mother for all first births to black and white women in the states of Washington, Louisiana, and Tennessee for the years 1976–1979, about 300,000 births in all. Looking down the first column of the table, we see that the NMR for 20–29 year olds is 48 percent lower than the NMR for mothers under age 20, which appears to support a policy of delay. Further examination suggests, however, that it would be a mistake to conclude from these data that encouraging women to time their first births in their 20s would substantially lower neonatal mortality rates.

Table 1. Neonatal Mortality Rates (NMRs) by Maternal Age and Race (deaths per 1,000 live births)

	All		Blacks		Whites	
Maternal age	NMR	Births (%)	NMR	Births (%)	NMR	Births (%)
<20	11.7	37	15.7	58	9.7	31
20–29	7.9	58	13.6	40	6.8	63
≥30	8.7	5	15.3	2	7.7	6
Total	9.3	100	14.9	100	7.8	100

Note: Data are from linked birth and infant death certificates for 305,907 first births in Washington, Louisiana, and Tennessee over the period 1976–1979.
Source: Calculated from Geronimus (1986a:table 1).

First, even if we attribute all of the differences in NMRs across maternal age groups to age per se, and even if *all* mothers were to postpone their first births until their 20s, the NMR would fall not by 48 percent but by 15 percent (9.3 overall vs. 7.9 in the 20s). More important, Table 1 illustrates that the differences between the NMRs for teen mothers and those in their 20s reflect differences in socioeconomic status (SES) across age groups. In particular, the NMR is much higher for blacks than for whites (overall and at any age), and a much higher proportion of blacks than whites have first births before age 20 (58 percent vs. 31 percent). As a result, looking across races biases (exaggerates) the relationship between maternal age and neonatal mortality. This is true even though race is a very crude control for SES: adding better controls would presumably attenuate the remaining differences between the teen and 20–29 NMRs within each race, since poor women, black and white, tend to have first births in their teens.

Because blacks have both high NMRs and a high percentage of teen births, a natural test of the efficacy of a policy of delay is to ask what the black NMR would be if black women were to follow some plausible pattern of postponed fertility, such as the white first-birth maternal age distribution. When standardized by the white distribution of maternal age at first birth (see Table 2), the overall black NMR falls only about 3 percent (from 14.9 to 14.4). This small change in the NMR would require a huge (almost 50 percent) reduction in the percentage of black first births to teenagers. Alternatively, we can approximate the effects of SES on NMRs by applying the white age-specific NMRs to the black maternal age schedule. This lowers the black NMR substantially, from 14.9 to 9.9 deaths per 1,000 births, or by about 34 percent. In sum, the evidence presented in these tables suggests that to reduce the U.S. NMR (hence the IMR), there is more to gain from policies that address issues of poverty and SES directly than by attempting to change the timing of births.

In this regard, it is noteworthy that Pampel and Pillai did not include increasing social welfare expenditures among their policy recommendations, even though their own results indicate that this could be an effective way to lower infant mortality. In particular, in regressions in which the infant mortality rate is the dependent variable, the unstandardized and the standardized coefficient on the ln(social welfare expenditures) variable is larger than the respective coefficient on the ln(teen births) variable (and is more highly statistically significant in one of two specifications for which a comparison is possible). As an illustration, one can calculate the approximate percentage change in each explanatory variable required to reduce the IMR by one point (holding the values of all other variables constant) simply by inverting the coefficients reported in Pampel and Pillai's table 3. To reduce the IMR by 1 death per 1,000 live births would require a 31–45 percent fall in the percentage of births to teenagers (all else held constant), whereas it would require a 13–18 percent rise in the share of GNP devoted to social welfare expenditures.[2] Evaluating these changes at the sample means reported in Pampel and Pillai's table 2, a 1 death per 1,000 drop in the infant mortality rate would require births to teens to fall from 7.1 percent to between 4.9 percent

Table 2. Standardized Black Neonatal Mortality Rates

Standardized by	Standardized NMR	% change in black NMR
White maternal age distribution	14.4	−3.3
White age-specific NMRs	9.9	−33.6

Note: The standardized NMRs are calculated by using the figures presented in Table 1. The first (14.4) results from multiplying the white maternal age distribution by the black age-specific NMRs and summing across age groups. The second (9.9) results from multiplying the black maternal age distribution by the white age-specific NMRs and summing. Percent change in NMR = 100 × (standardized NMR − actual NMR)/actual NMR.

and 3.9 percent of all births, or it would require an increase in social welfare expenditures from 10.9 percent to between 12.3 percent and 12.9 percent of GNP. Moreover, the social welfare measure used by Pampel and Pillai is very broad; for example, it includes social security payments. Presumably a much *smaller* increase in spending on social welfare programs specifically targeted to poor mothers and children (such as AFDC or WIC) could achieve the same result.

Notes

[1] Although the variables included in Pampel and Pillai's regressions capture some of the intertemporal and cross-country variation in poverty [e.g., per capita gross national product (GNP), the Gini coefficient, and the unemployment rate], they are likely to leave much of poverty unmeasured. What is more important for the issue of infant mortality, they leave much of poverty *births* unmeasured. The Gini coefficient is a measure of overall income inequality, not of (absolute) poverty per se. Moreover, due to data limitations, Pampel and Pillai included only one Gini coefficient for each country (rather than one each period for each country) and used outside regression estimates to impute values for Ireland and Belgium. Similarly, per capita GNP is not necessarily correlated with the size of the poverty population within and across developed countries (e.g., the U.S. has both a very high level of per capita GNP and a large poverty population compared with other developed nations). Finally, the unemployment rate (the percentage of the labor force not currently employed) excludes both the working poor and those poor people who are not counted as part of the labor force and includes the non-poor unemployed. Our point is simply that even though these are important control variables, the teen births variable may nonetheless be picking up the effects of poverty births.

[2] The regression reported in column 9 of Pampel and Pillai's (1986) table 3 does not include the welfare expenditure variable, so we cannot directly compare coefficients. The effect of teen births is, however, much smaller in this specification, indicating that a 4,500% reduction in teen births would be needed to reduce the infant mortality rate by 1 death per 1,000 births.

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