

Black-White Differences in Maternal Age, Maternal Birth Cohort, and Period Effects on Infant
Mortality in the U.S. (1983-2002)

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Abstract

We investigate three interrelated sources of change in infant mortality rates over a 20 year period using the National Center for Health Statistics (NCHS) linked birth and infant death cohort files. The effects of maternal age, maternal birth cohort, and time period of childbirth on infant mortality are estimated using a modified age/period/cohort (APC) model that identifies age, period, cohort effects. We document black-white differences in the patterning of these effects. We find that maternal age effects follow the predictable U-shaped pattern, net of period and cohort, but with a less steep gradient in the black population. The largest relative maternal age-specific disparity in IMR occurs among older African American mothers. Cohort effects, while considerably smaller than age and period effects, present an interesting pattern of a modest decline in IMR among later cohorts of African American mothers coupled with an increasing IMR among the same cohorts of non-Hispanic whites.

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Introduction

Temporal change in infant mortality involves period-driven technological advances in the face of changing childbearing contexts experienced by different birth cohorts of mothers, in combination with changes in the maternal age patterns of childbearing. We argue that an age-period-cohort (APC) analysis could prove useful to disentangle these interrelated effects in a study of infant mortality, and that this kind of analysis can provide insight into the temporal components of black-white differences in infant mortality rates. In particular, a cohort analysis can reveal differences that would not be revealed by consideration of age and period alone, or by approaches that fail to account for inherent dependence of these temporal dimensions.

APC analysis is a well known approach to gain insight into the unique contribution of age, birth cohort, and time period in mortality research. Age typically represents variation associated with different age groups and attributable to differences in physiological changes, life experiences and changes in social roles. Period effects represent variation over time that simultaneously affects all age groups. Cohort effects represent variation associated with groups of individuals having different formative experiences at successive ages in successive years. A common goal is to assess the effects of one of these temporal dimensions net of the effects of the other two (Yang et al. 2008).

With regard to mortality processes, biological age accounts for considerable variation. Yet, some of this variation is shaped by the historical period in which death occurs and by a cohort's unique experience of history. Although the most straightforward application of APC is in the analysis of adult

mortality, the APC analytic approach may offer insights about aspects of infant mortality, which have not thus far been explored in past research. In particular, the historical time period in which infant death occurs, the age of the mother at the time of childbirth, and the mother's birth cohort may play important distinct roles in infant mortality. As such, maternal age, mother's birth cohort, and time period of infant death potentially tap distinct dimensions of change which are not easily separable due to the perfect linear dependence of mother's birth cohort on maternal age and time period in which infant death occurs.

Background

Infant mortality rates have declined considerably over the past 20 years. We can attribute some share of the decline to medical innovations targeting specific leading underlying causes of infant death, such as the high-technological innovations responsible for the reduction in mortality associated with leading causes of infant death—such as pre-natal screening in the case of congenital anomalies and surfactant replacement therapy in the case of respiratory distress syndrome—as well as and low-technological innovations (i.e., the “back to sleep” campaign) associated with reduction in mortality due to SIDS (Powers and Song 2009). It seems reasonable that interventions of this type would be captured by period effects in an Age-Period-Cohort (APC) analysis. Additionally, we would expect that some portion of the decline in infant mortality is impacted by the changing maternal age structure of childbearing, perhaps affected by offsetting trends toward older ages of childbearing and changes in teenage childbearing rates. This component of change would be captured by age effects in an APC analysis (specifically maternal age effects).

A third temporal component reflects secular changes in the experience of various cohorts of women. Cohort effects may tap the tendency toward smaller families in later birth cohorts or may reflect

secular trends in teen pregnancy rates or may reflect distributional changes in the correlates of mortality such as education. The extent that these components of change are mirrored across distinct populations is also of interest. For example, later birth cohorts of African American women may have benefitted in important ways from the Civil Rights movement and the accompanying changes in racial attitudes and the change in the degree of discrimination towards racial and ethnic minorities in ways that non-Hispanic white women did not, whereas both the African American and non-Hispanic white populations would be expected to benefit by cohort change in the distribution of education. A comparative analysis by race could be informative in isolating these cohort effects, net of changes in the maternal age distribution and with respect to the historical period in which childbearing and infant mortality occurs.

Data and Methods

The NCHS linked birth and infant death cohort files from 1983-2002 provide a series of data consisting of millions of births per year, with an exceptional match rate. About 98% of the infant death records were successfully linked to birth certificates in any given year (U.S. Department of Health and Human Services 1995). However, no linked birth/infant death cohort files were produced by the NCHS from 1992-94. The resulting study population consists of individual-level records for 62,878,862 live births and 530,635 infant deaths. As is customary in this research, race of mother, as indicated on the birth certificate is used to ascertain race of infant. This yields an analytic sample of 10,054,428 births and 152,793 infant deaths in the non-Hispanic black population and 51,767,843 births and 351,221 infant deaths in the non-Hispanic white population.

We carry out an APC analysis of the NCHS cohort-linked birth death files from 1983 to 2002 to examine infant mortality using information on mother's age (reported on the birth certificate) and year of infant death. Data are arranged in seven 5-year maternal age groupings (A) ranging from age 15 or

less to age 45 and older, as well as four 4-year periods (P) spanning the period from 1983 to 2002.¹ This yields ten 5-year maternal birth cohorts ranging from 1940 to 1985. It should be noted that certain cohorts might be incompletely represented as the NCHS linked file data are not available from 1992-1994. Most problematic would be the failure to capture the relatively few later births that occurred to earlier cohorts during this period.

These data adhere to the assumptions of APC analysis insofar as mother's birth cohort (C) is linearly dependent on period and age $C = P - A$. As is well known, data in this form suffer from an identification problem that renders conventional estimation of all the age-period-cohort effects impossible without imposing constraints on at least 1 of the model parameters. Yang et al. (2004) proposed an estimator that yields estimates of all quantities that we implement here. We also examine some alternative approaches.

Descriptive Summary

Tables 1 and 2 present the data arrayed by maternal age and maternal birth cohort in terms of number of births and infant mortality rates for non-Hispanic whites and non-Hispanic blacks in the NCHS data. Figure 1 shows the smoothed empirical IMRs by maternal age and year for each group along with the black/white rate ratios using a finer measurement scale than was used in Tables 1 and in the analytic models discussed later. Maternal Birth cohort specific rates are reflected along the diagonals of Figure 1. We notice somewhat different maternal age profiles of risk depending on racial group, with non-Hispanic whites exhibiting markedly steeper infant mortality gradients at younger and

¹ The age and period groupings necessarily affect the number of maternal birth cohorts that would be generated in an APC analysis. Given the limitations of the data collection for the years from 1991-1994, we have used the following period groupings and coding in order to ensure adequate cell sizes: 1983-1985=1985 1986-1990=1990 1991-1998=1995 1999-2002=2000. When combined with the 5 year age classification, this yields the following cohort classification and coding: 1935-1939=1940, 1940-1944=1945, 1945-1949=1950, 1950-1954=1955, 1955-1959=1960, 1960-1964=1965, 1965-1969=1970, 1970-1974=1975, 1975-1979=1980, 1980-1984+ =1985.

older ages than non-Hispanic blacks. Each group experiences a flattening of the generally U-shaped pattern of infant mortality associated with maternal age. We also observe a somewhat more dramatic absolute reduction in infant mortality over time among non-Hispanic blacks when compared to non-Hispanic whites. However, the empirical black/white rate ratios in the rightmost panel of Figure 1 demonstrate the well known increasing relative black-white disparities occurring throughout the past several decades (see e.g., Frisbie et al. 2004). However, the 3-dimensional depiction by maternal-age and time period reveals that this widening relative disparity is quite obvious over the 25-35 maternal age range and less obvious for women under 20 and over 35. The two leftmost panels of Figure 1 suggest that the source of this relative increase is the combination of steeper declines in maternal age specific infant mortality among non-Hispanic whites in later decades coupled with a stagnating decline from the mid-1990s to 2002 among non-Hispanic blacks.

APC Model

The APC model conditions on the marginal distributions of age and period. The cohort distribution may be viewed as an age \times period interaction along the diagonals of an age by period contingency table. In a conventional generalized linear model (glm) model, the linear dependence of the cohort effects requires equating two model coefficients. In this case, the usual dummy-variable constraints are used to identify the remaining age and period effects. Alternatively, one could constrain age or period effects in the same manner and estimate all but two of the cohort effects. We can rule out constrained models through a series of likelihood ratio tests on all possible two-way models shown below. The preferred model for both racial groups based on several fit criteria is the full APC model.

Non-Hispanic Whites							
Model	Obs	ll(null)	ll(model)	df	AIC	BIC	
AP	28	.	-180.6805	10	381.3609	394.6829	
AC	28	.	-402.8235	16	837.647	858.9623	
PC	28	.	-2515.107	13	5056.214	5073.532	
APC	28	.	-162.8175	18	361.635	385.6146	

Non-Hispanic Blacks							
Model	Obs	ll(null)	ll(model)	df	AIC	BIC	
AP	28	.	-156.3352	10	332.6705	345.9925	
AC	28	.	-236.5511	16	505.1022	526.4174	
PC	28	.	-292.1949	13	610.3897	627.7084	
APC	28	.	-142.2278	18	320.4556	344.4352	

Although these tests provide evidence that the full APC model is preferred, these results do not rule out the existence of plausible constraints on one or more APC parameters that would lead to a simpler model. These types of constraints generally require external information for justification, i.e., beyond simply an empirical examination.

The APC model is specified as

$$\log E(r_{ij}) = \log E\left(\frac{d_{ij}}{n_{ij}}\right) = \beta_0 + \beta_i^A + \beta_j^P + \beta_k^C, \quad (1)$$

where $\log E(r_{ij})$ is the logarithm of the expected IMR based on d_{ij} deaths and n_{ij} births pertaining to cell ij of a cross-classification of infant deaths and births in maternal age interval i (for $i = 1, \dots, I$ age groups) and time period j (for $j = 1, \dots, J$ periods). Age and period effects are denoted by β_i^A and β_j^P , respectively. β_k^C denotes the k th (diagonal) maternal birth cohort effect (for $k = 1, \dots, I + J - 1$ birth cohorts), and where the index $k = I - i + j$. For this study, $I = 7, J = 4$ for $N = I \times J = 28$ age by period cells covering 10 maternal birth cohorts.

The following ANOVA, centered-effects (or sigma constraint) normalization is imposed on the effects $\sum \beta_i^A = \sum \beta_j^P = \sum \beta_k^C = 0$. Alternatively, the model can be cast terms of multiplicative effects,

$$E(r_{ij}) = \tau_0 \tau_i^A \tau_j^P \tau_k^C, \quad (2)$$

where $\prod \tau_i^A = \prod \tau_j^P = \prod \tau_k^C = 1$. The τ parameters under the APC model are multiplicative effects whose product is 1 over the levels of each factor. This normalization ensures that the constant term in the model (τ_0) is the scaled grand mean IMR (or central IMR). The APC estimates therefore reflect the maternal age, maternal birth cohort and time period departures from the central mean IMR for each population, net of the other APC effects.

Estimating the APC Model

Yang et al. (2004) propose an estimator that decomposes the less-than full-rank design matrix to provide a solution to the APC identification problem via principal components regression. This yields the so-called “intrinsic estimator” (IE). Although we refer to the IE as an “estimator” it is more appropriately viewed as an estimable function. We forego the description of the underlying vector geometry of the solution for the IE—which is described elsewhere—and focus on the mechanics of estimation. The IE is easily implemented for a linear model using well known procedures for dealing with ill-conditioning arising from collinear predictors, which involve finding characteristic roots and orthonormal vectors that are linear transformations of the model design matrix. Formally, Let \mathbf{Q} be the $p \times p$ orthogonal matrix of eigenvectors of the $\mathbf{X}'\mathbf{X}$ matrix based on the $N \times p$ design matrix \mathbf{X} composed of an intercept term, $I - 1$ age terms, $J - 1$ period terms, and $I + J - 2$ cohort terms subject to the ANOVA normalization above. Let ℓ_1, \dots, ℓ_p denote the eigenvalues of $\mathbf{X}'\mathbf{X}$ and \mathbf{L} be the $p \times p$ diagonal matrix containing these eigenvalues. Then, because $\mathbf{QLQ}' = \mathbf{X}'\mathbf{X}$, the IE can be obtained as the solution to the following principle components regression

$$\mathbf{b}_{\text{IE}} = (\mathbf{QL}_0^{-1}\mathbf{Q}')(\mathbf{X}'\mathbf{y}), \quad (3)$$

where \mathbf{y} is the $N \times 1$ response vector—which is typically in the form of log rates—and \mathbf{L}_0^{-1} is the $p \times p$ diagonal matrix containing $\ell_1^{-1}, \dots, \ell_{p-1}^{-1}, 0$ on the main diagonal and zeros elsewhere. In this case $\mathbf{Q}\mathbf{L}_0^{-1}\mathbf{Q}'$ plays the role of $[\mathbf{X}'\mathbf{X}]^{-1}$ in the usual linear regression model.

Yang et al.'s 2004 approach is one of several strategies to obtain unique estimates from under-identified models, of which the APC model is a special case. More generally, we can view the IE as the limit of a coefficient vector from a penalized regression where the shrinkage penalty $\lambda \rightarrow 0^+$ (see e.g., Fu 2000), in which case we may dispense with the step of computing eigenvalues/vectors and work directly with \mathbf{X} . The estimator then takes the form of a ridge regression (Hoerl 1962; Hoerl and Kennard 1970)

$$\mathbf{b}_R = (\mathbf{X}'\mathbf{X} + \lambda\mathbf{I})^{-1}(\mathbf{X}'\mathbf{y}), \quad (4)$$

where \mathbf{I} is the $p \times p$ identity matrix. This model may also be motivated from a Bayesian perspective in which case prior data \mathbf{X}_0 and \mathbf{Y}_0 is brought to bear on the estimation. In this case, $\mathbf{Y}_0 = \mathbf{0}$, $\mathbf{X}_0'\mathbf{X}_0 = \mathbf{I}$ and $\lambda \rightarrow 0^+$ constitutes non-informative prior information (Marquardt 1970; Draper and Smith 1981). It is well known that the ridge estimator is biased. However, there is a tradeoff between bias and variance, with the variance in the ridge estimator decreasing as $\lambda \rightarrow \infty$. Typically, a cross-validation strategy is employed to find the optimal λ value for a particular set of data. The resulting standard errors of \mathbf{b}_R when $\lambda > 0$ are smaller than those of \mathbf{b}_{IE} (i.e., \mathbf{b}_R has lower mean square error than \mathbf{b}_{IE}). In applied work, one may use the ridge estimator for APC analysis and set λ to a very small number (i.e., 1.e-8) so that $\mathbf{b}_R \approx \mathbf{b}_{IE}$. However, cross-validation may reveal a more suitable λ for the data.

Finally, perhaps less “mysterious” than implied by Yang et al.’s 2004 exposition, the IE may be computed using an OLS estimator that employs a “pseudo”, Moore-Penrose, or generalized inverse of $\mathbf{X}'\mathbf{X}$ denoted by $(\mathbf{X}'\mathbf{X})^+$ (Searle 1971; Marquardt 1970),

$$\mathbf{b}_{IE} = (\mathbf{X}'\mathbf{X})^+(\mathbf{X}'\mathbf{y}). \quad (5)$$

Employing the generalized inverse obviates computing eigenvalues/vectors as these steps are subsumed in the generalized inverse (Marquardt 1970). In summary, a number of equivalent numerical methods (i.e., principal components, singular value decomposition, generalized inverse, etc.) may be employed in various ways to provide solutions to problems where the design matrix is rank deficient (i.e., $\mathbf{X}'\mathbf{X}$ is ill-conditioned). These methods are not new, and date back at least 50 years. In the case of APC analysis, the problem is simplified because there is only one collinear predictor (i.e., one too many columns in the design matrix) yielding a single zero eigenvalue of $\mathbf{X}'\mathbf{X}$, which gives \mathbf{X} an effective rank of $p-1$.

Maximum Likelihood Estimation

Least squares-type solutions outlined above require one matrix inverse operation. In the case of the generalized linear models used here (i.e., loglinear models for rates based on counts of deaths and exposure to risk), we require iterative updating of the estimates in order to maximize a log likelihood function of the form

$$\log L = \sum_{i=1}^N d_i \mathbf{X}_i \boldsymbol{\beta}_{ML} - n_i e^{\mathbf{X}_i \boldsymbol{\beta}_{ML}}, \quad (6)$$

where d and n pertain to the number of deaths and exposures in each age by period “cell,” respectively. The most straightforward optimization approach employs the Moore-Penrose inverse to the matrix of second derivatives of $\log L$ with respect to $\boldsymbol{\beta}_{ML}$ in Eq. (6) in the updating equations for a generalized linear model using a Newton-Raphson algorithm, where at iteration $t + 1$, the estimator is updated as

$$\mathbf{b}_{\text{ML}}^{t+1} = \mathbf{b}_{\text{ML}}^t - [\mathbf{H}^t]^+ \mathbf{g}^t, \quad (7)$$

where \mathbf{g}^t is the $p \times 1$ vector of 1st derivatives of the log likelihood function with respect to the APC parameters (i.e., the gradient), \mathbf{H}^t is the $p \times p$ matrix of 2nd derivatives (i.e., the Hessian), each of which is evaluated at iteration t . That is, in a nonlinear estimation \mathbf{H} assumes the role that $\mathbf{X}'\mathbf{X}$ plays in the linear case (Marquardt 1970).

As mentioned earlier, the results from the model adopted here are normalized using an ANOVA-type or “centered-effects” coding, thus providing estimates for *all* the APC effects and the grand mean (or central log rate). Like dummy variable regression, an omitted category must be chosen to identify the model. Under the sigma constraint normalization, this effect is simply minus the sum of the effects of the included categories for a particular APC factor. Thus, obtaining the omitted category effects is straightforward. Obtaining their standard errors is a technical aspect of the model that is not well documented, so we discuss this in some detail. Using the last level of each APC factor as the omitted category, we obtain the excluded category effects as follows:

$$b_i^A = -\sum_{i=1}^{I-1} b_i^A, \quad b_j^P = -\sum_{j=1}^{J-1} b_j^P, \quad \text{and} \quad b_{I+J-1}^C = -\sum_{k=1}^{I+J-2} b_k^C. \quad (8)$$

Following the usual rule for the variance of a sum, the variances of the omitted category effects may be computed as

$$\text{var}(b_i^A) = \sum_{j=1}^{I-1} \sum_{k=1}^{I-1} \text{cov}(b_{jk}^A), \quad \text{var}(b_j^P) = \sum_{j=1}^{J-1} \sum_{k=1}^{J-1} \text{cov}(b_{jk}^P), \quad \text{and} \quad \text{var}(b_{I+J-1}^C) = \sum_{j=1}^{I+J-2} \sum_{k=1}^{I+J-2} \text{cov}(b_{jk}^C), \quad (9)$$

where $\text{cov}(b_{jk}^{\text{APC}})$ denotes element $j k$ of the variance/covariance matrix pertaining to the APC effects, and are obtained at the final optimization iteration as $\text{cov}(\mathbf{b}_{\text{ML}}) = [-\mathbf{H}]^+$.

Results from the APC Model

Combining relevant model terms, we generate the maternal age, maternal birth cohort and period specific IMRs as shown in Figures 2 and 3 from each model as $\hat{\tau}_0 \hat{\tau}_i^A \hat{\tau}_j^P \hat{\tau}_k^C \times 1,000$. The central mean IMR—denoted by the dotted line appearing through each plot in Figure 2—is $\hat{\tau}_0$. We can see for example, that the estimated IMR in the population of women in the under age 15 and over 40 age groups is higher than the grand mean IMR, conditional on period and cohort. This pattern is consistent with the U-shaped infant mortality pattern, with relatively higher infant mortality associated with both younger and older maternal ages. Figure 2 shows a steeper age gradient in IMR in the non-Hispanic white population and a somewhat steeper period gradient in the non-Hispanic black population, which is also consistent with the empirical patterns given in Figure 1. The lower maternal age gradient in the black population is well known and consistent with the conceptual framework of weathering (Geronimus 1992). Consistent with Figure 1, Figure 3 shows that the largest relative black-white infant mortality gap occurs for women aged 30-40.

Figure 2 also shows that period effects are markedly monotonically decreasing in each population. The span of the reduction among non-Hispanic blacks is on the order of 5.37 infant deaths per 1,000 births compared to a reduction of 4.78 deaths per 1,000 births among non-Hispanic whites. Thus, in an absolute sense, conditional on mother's cohort and age, a somewhat larger decline is observed in the non-Hispanic black population. This tendency runs counter to the observed pattern in Figure 1, which shows a somewhat steeper period gradient in the non-Hispanic black population. We may attribute this component to technological changes occurring during the time period under study. However, we must conclude that these changes appear to have about equal impact on the decline in infant mortality for both non-Hispanic whites and non-Hispanic blacks.

Cohort change in IMR is much less pronounced. However, the cohort patterns are considerably more interesting from a comparative standpoint. Among non-Hispanic whites, there is a moderate increase in IMR by birth cohort, with evidence of increasing infant mortality in later cohorts of mothers, net of maternal age and period. There is somewhat less precision in the estimates for the oldest and youngest cohorts, particularly in the non-Hispanic black population. Nevertheless, patterns of cohort change in infant mortality among non-Hispanic blacks are notably distinct from those of non-Hispanic whites. Net of changes in the maternal age-specific rates and period-specific rates, cohort change in IMR among non-Hispanic blacks reveals a moderate shift toward lower infant mortality for younger cohorts of African American women. This pattern is in sharp contrast to the cohort patterns evident among non-Hispanic whites, where earlier cohorts of women experience relatively lower infant mortality in contrast to more recent cohorts. The fact that a main portion of cohort decline in IMR occurs for the population of African American women born after the civil rights movement leads us to speculate that a modest survival benefit accrued to the infants whose mothers were the likely beneficiaries of positive social change occurring in this period. However, this benefit is overwhelmingly overshadowed by period changes.

Figure 4 shows the black/white age, period, and cohort specific rate ratios based on the APC specific rates for each population. We calculate the age, period, cohort specific rates from each model as $\hat{r}_{ijk} = \hat{\tau}_0 \hat{\tau}_i^A \hat{\tau}_j^P \hat{\tau}_k^C$ then form the ratio of the black and white rates. The dotted vertical line appearing in the Figure 4 depicts the central black/white rate ratio of 1.93, from which the other rate ratios depart. This may be viewed as an “expected” relative disparity of about 2 infant deaths in the black population for each infant death in the white population, evaluated at the average age at birth, time period, and maternal birth cohort. Rate ratios appearing on the left of the line denote smaller than expected relative black/white disparities in specific APC effects, whereas those to the right indicate larger than expected

disparities in specific APC effects. Relative disparities in IMR by maternal age exhibit narrower than expected black/white gaps at younger maternal ages, a widening black/white relative gap over the prime childbearing years (25-35), and moderate narrowing at later ages. Period effects show the increasing relative black/white gaps over the 20-year period. Both the age and period effects mirror the empirical depiction of Figure 1. More interestingly, consistent with earlier results, cohort effects exhibit narrowing of the relative black/white gap for later cohorts of black women. Figures 2 and 3 suggest that this is due to higher infant mortality experienced in later cohorts (1965 and onward) of non-Hispanic white mothers rather than to the relatively modest declines in infant mortality that occurred among the same cohorts of non-Hispanic black women.

Thus far we have considered results based on predicted rates from the APC models. We now compare the estimated APC effects by group and assess their relative magnitude. These results permit a direct comparison of race-specific APC effects. The top panel (a) of Figure 5 shows the age, period and cohort effects (b^A , b^P , and b^C) for each group plotted against age, period and cohort respectively. The lower panel (b) of Figure 5 shows the black/white relative risk ratios: $e^{b_B^A} / e^{b_W^A}$, $e^{b_B^P} / e^{b_W^P}$, and $e^{b_B^C} / e^{b_W^C}$.

We see from panel (a) that the maternal age effects for non-Hispanic whites exhibit more variation when compared to non-Hispanic blacks and reveal both higher effects at younger maternal ages and lower effects in the prime childbearing years when compared to non-Hispanic blacks. This is highlighted in panel (b) which shows that maternal age effects for non-Hispanic blacks are relatively lower than those of whites at younger ages and relatively higher in the prime childbearing years. Period effects show similar patterns but are somewhat stronger for non-Hispanic whites. A modest crossover is evident in the relative effect (i.e., the period effect for blacks is initially lower from 1983 to 1990 and then moderately higher thereafter). Period effects show modest increases for whites coupled with a level tendency and moderate decline for blacks. The relative differences in these effects reveal a clearer

picture of a modest infant mortality advantage for cohorts of African women born after 1965. However, given the flat profile of cohort effects for non-Hispanic blacks, this partially reflects an infant mortality disadvantage accruing to non-Hispanic whites born after 1975.

Summary

This paper applies APC modeling to disentangle maternal age, time period and maternal birth cohort effects in 17 years of cross-sectional longitudinal data on infant mortality in the U.S. based on the NCHS linked birth-infant death files from 1983 to 2002. While it is common to apply APC analysis to adult mortality where age at death, time period of death, and birth cohort are the relevant interrelated temporal dimensions, this paper is the first that we are aware of to apply an APC analytic strategy to the study of infant mortality and to the study of white-black racial disparities in infant mortality. In this case, time period is the year that infant death occurs, while in any given year this event can be experienced by different birth cohorts of U.S. women and at different points in the life course. The estimation approach is able to uniquely identify the separate components of change in IMR over 2 decades. We find that, while period effects dominate change in infant mortality over this time frame, cohort effects reveal a modest closing of the absolute black-white infant mortality gap. We find that while some narrowing of the gap may be attributable to lower mortality occurring to non-Hispanic black infants whose mothers' grew up in the post-civil rights era, a larger contributor to the narrowing gap appears to be the increasing infant mortality experienced by later cohorts of non-Hispanic whites.

Understanding the sources of these changes will require additional data, beyond the 3 measures used here. In particular, a fruitful area of further research would be to compare the black-white differentials by level of education, as this may reveal potentially interesting cohort differentials. In this case, separate APC model would be fit to each population within each educational stratum, yielding

APC effects that are conditional on race and education. A noteworthy limitation of the foregoing analysis is the failure to control for a number of confounding factors that may alter the associations between infant mortality and maternal age, time period and cohort. While it is “statistically” straightforward to include controls in the APC specifications described above as components of the design matrix, the APC specification adopted here is less flexible than hierarchical APC models which break the linear dependency by treating period and cohort as crossed random effects at level-2 nested in age at level-1 (Yang and Land 2008). Generalized linear mixed models and Bayesian simulation (Markov-Chain-Monte-Carlo [MCMC]) methods are the widely recommended choice when fitting multivariate APC models as they allow mediation of cohort and period specific effects through the inclusion of relevant predictors in the level-2 submodels, and permit flexible specifications of fixed effects in the level-1 submodel. Finally, in addition to the substantive findings presented here, we hope to show that there are a variety of analytical methods for dealing with singular design as is characteristic of APC analysis. The IE, while seemingly offering something new and improved, has its origins in methodologies developed over a half century ago.

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Table 1: Number of live births and infant deaths by maternal age and maternal birth cohort among Non-Hispanic whites (NCHS cohort linked files).

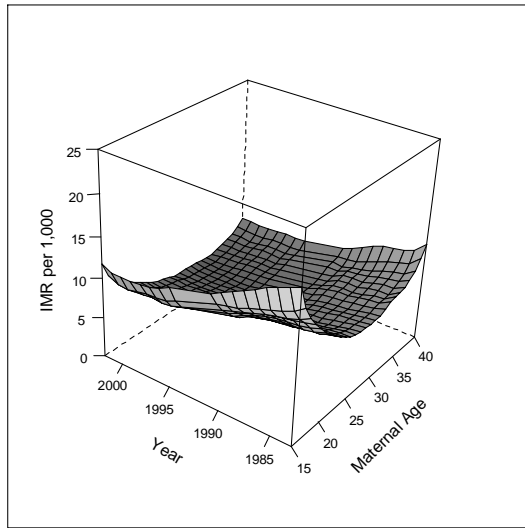
Live Births								
Non-Hispanic Whites								
Maternal Age								
Maternal Birth Cohort	<15	15-19	20-24	25-29	30-34	35-39	40+	Total
1940							64,741	64,741
1945						474,653	159,806	634,459
1950					1,635,236	1,096,974	96,954	2,829,164
1955				2,899,837	3,299,577	577,166	486,641	7,263,221
1960			2,690,494	5,044,245	1,465,898	2,426,426		11,627,063
1965		966,368	4,099,337	1,840,702	5,175,650			12,082,057
1970	11,709	1,609,930	1,542,453	5,956,100				9,120,192
1975	21,115	686,381	5,139,852					5,847,348
1980	10,662	2,257,686						2,268,348
1985	31,252							31,252
Total	74,738	5,520,365	13,472,136	15,740,884	11,576,361	4,575,219	808,142	51,767,845

Infant Deaths								
Maternal Age								
Maternal Birth Cohort	<15	15-19	20-24	25-29	30-34	35-39	40+	Total
1940							722	722
1945						3,980	1,534	5,514
1950					12,138	7,888	713	20,739
1955				21,643	21,502	3,498	3,362	50,005
1960			25,153	33,073	7,684	12,782		78,692
1965		12,425	34,274	9,981	23,210			79,890
1970	261	18,598	10,868	28,665				58,392
1975	395	6,535	31,398					38,328
1980	166	18,314						18,480
1985	459							459
Total	1,281	55,872	101,693	93,362	64,534	28,148	6,331	351,221

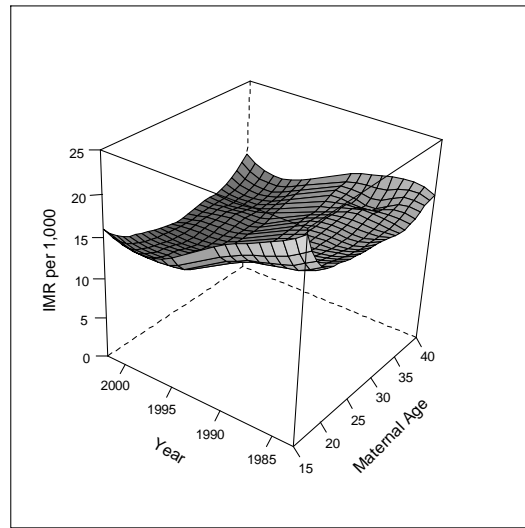
Table 2: Number of live births and infant deaths by maternal age and maternal birth cohort among Non-Hispanic blacks (NCHS cohort linked files).

Live Births								
Non-Hispanic Blacks								
Maternal Age								
Maternal Birth Cohort	<15	15-19	20-24	25-29	30-34	35-39	40+	Total
1940							11,249	11,249
1945						65,950	23,495	89,445
1950					203,251	148,055	13,531	364,837
1955				406,179	427,145	76,383	68,642	978,349
1960			570,353	766,879	187,795	314,418		1,839,445
1965		383,154	1,012,102	285,051	629,935			2,310,242
1970	16,301	685,136	388,456	925,853				2,015,746
1975	29,599	274,746	1,292,597					1,596,942
1980	11,846	809,046						820,892
1985	27,281							27,281
Total	85,027	2,152,082	3,263,508	2,383,962	1,448,126	604,806	116,917	10,054,428

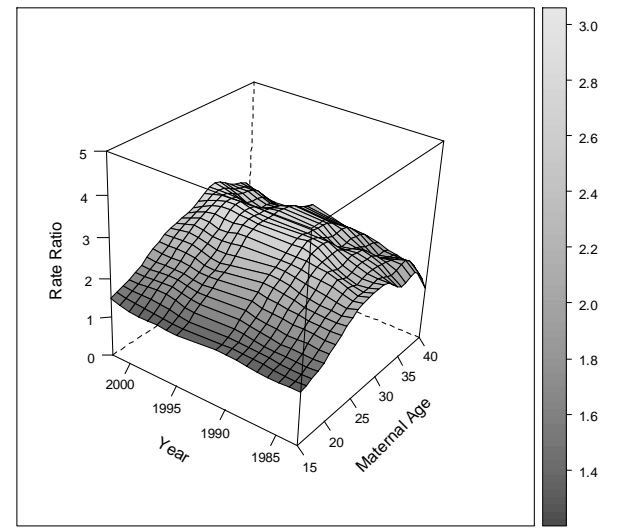
Infant Deaths								
Maternal Age								
Maternal Birth Cohort	<15	15-19	20-24	25-29	30-34	35-39	40+	Total
1940							203	203
1945						1,183	449	1,632
1950					3,445	2,496	236	6,177
1955				6,884	7,083	1,209	1,022	16,198
1960			10,267	12,576	2,743	4,127		29,713
1965		7,462	17,047	4,012	8,000			36,521
1970	437	12,119	5,700	11,456				29,712
1975	703	4,074	16,305					21,082
1980	249	10,819						11,068
1985	487							487
Total	1,876	34,474	49,319	34,928	21,271	9,015	1,910	152,793



Non-Hispanic White



Non-Hispanic Black



Black/White Rate Ratio

Figure 1: IMR per 1,000 by period, maternal age and race and black/white rate ratios (smoothed estimates). The maternal age label 15 denotes the interval [< 15) and the maternal age label of 40 denotes the interval [$40+$), otherwise age is measured in single years.

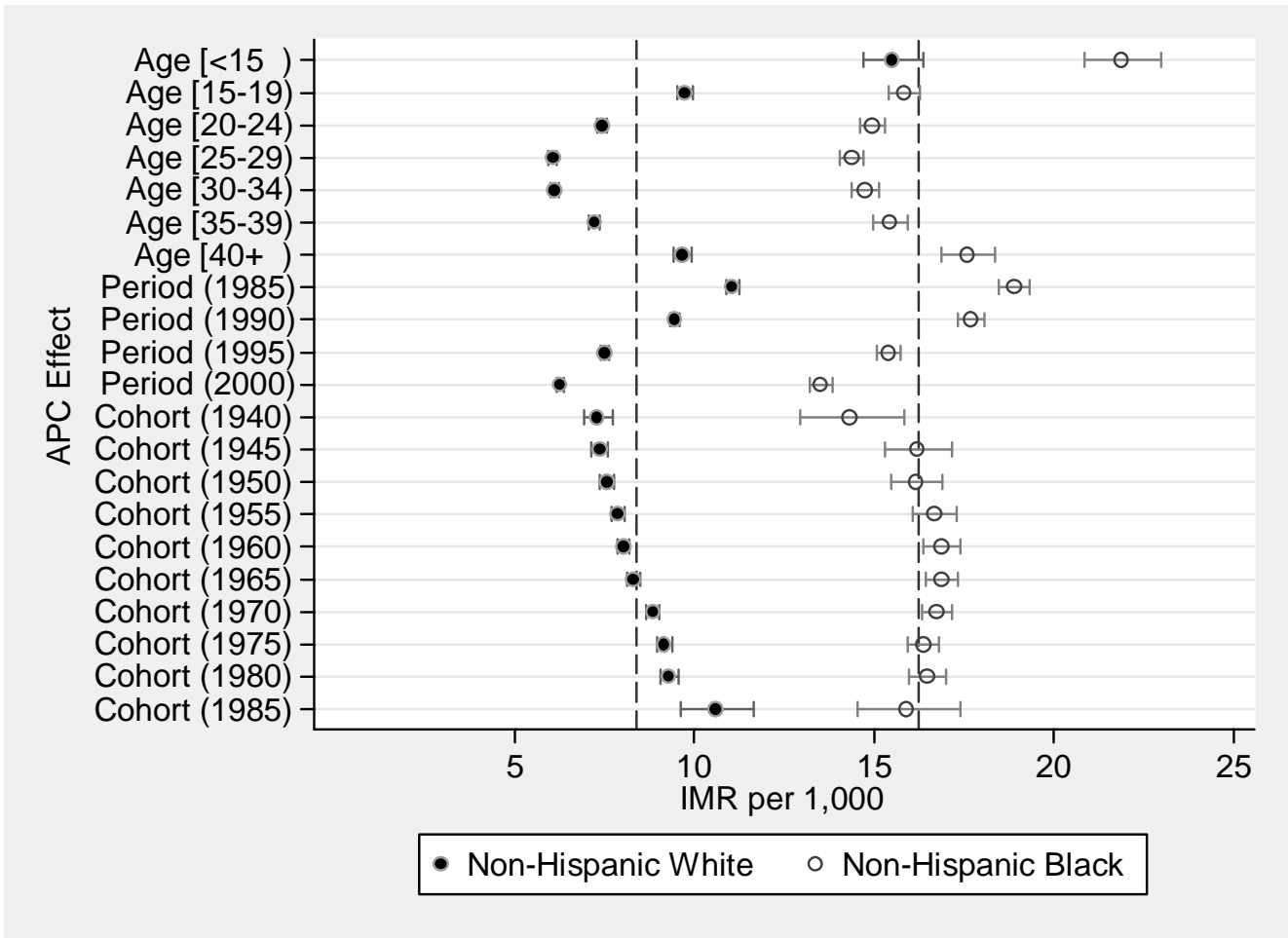


Figure 2: Interval estimates of maternal age, period, and maternal birth cohort specific infant mortality rates (per 1,000 live births) for non-Hispanic whites and blacks. The dashed lines are the central IMRs for each population.

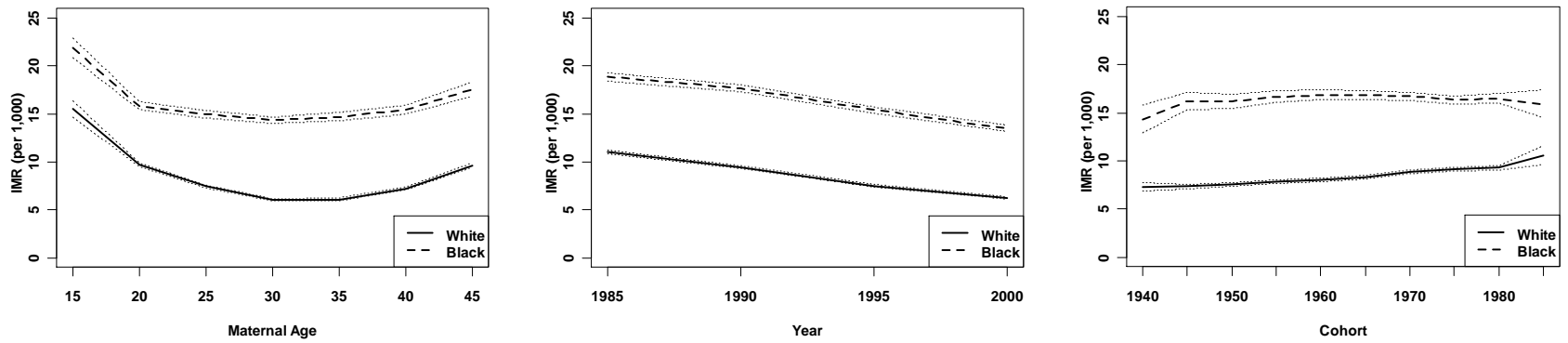


Figure 3: Estimated material age, period, and maternal birth cohort specific infant mortality rates (per 1,000 live births) for non-Hispanic whites and non-Hispanic blacks (with 95% confidence intervals).

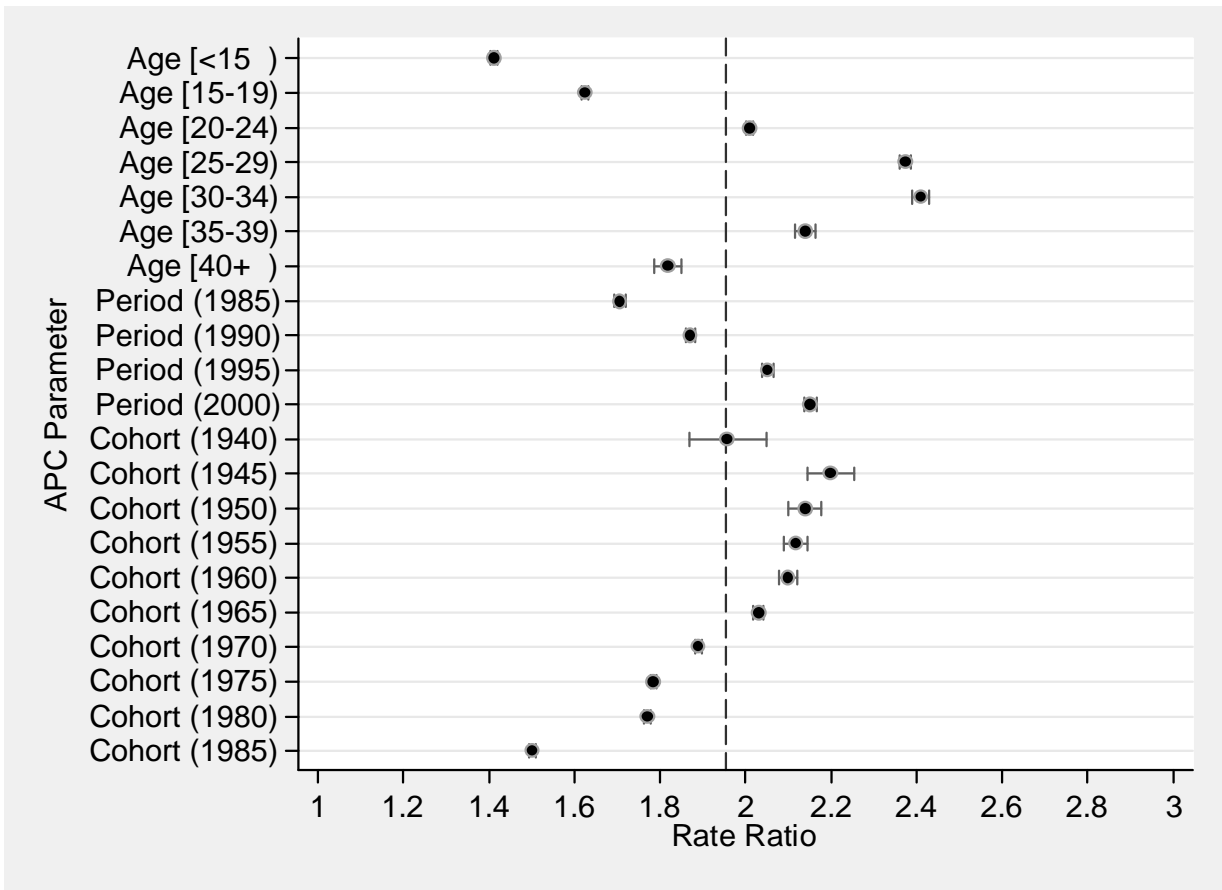


Figure 4: Interval estimates of black/white rate ratios quantifying the relative infant mortality disparities in Figure 2. The vertical dashed line is the black/white “central” rate ratio of 1.93.

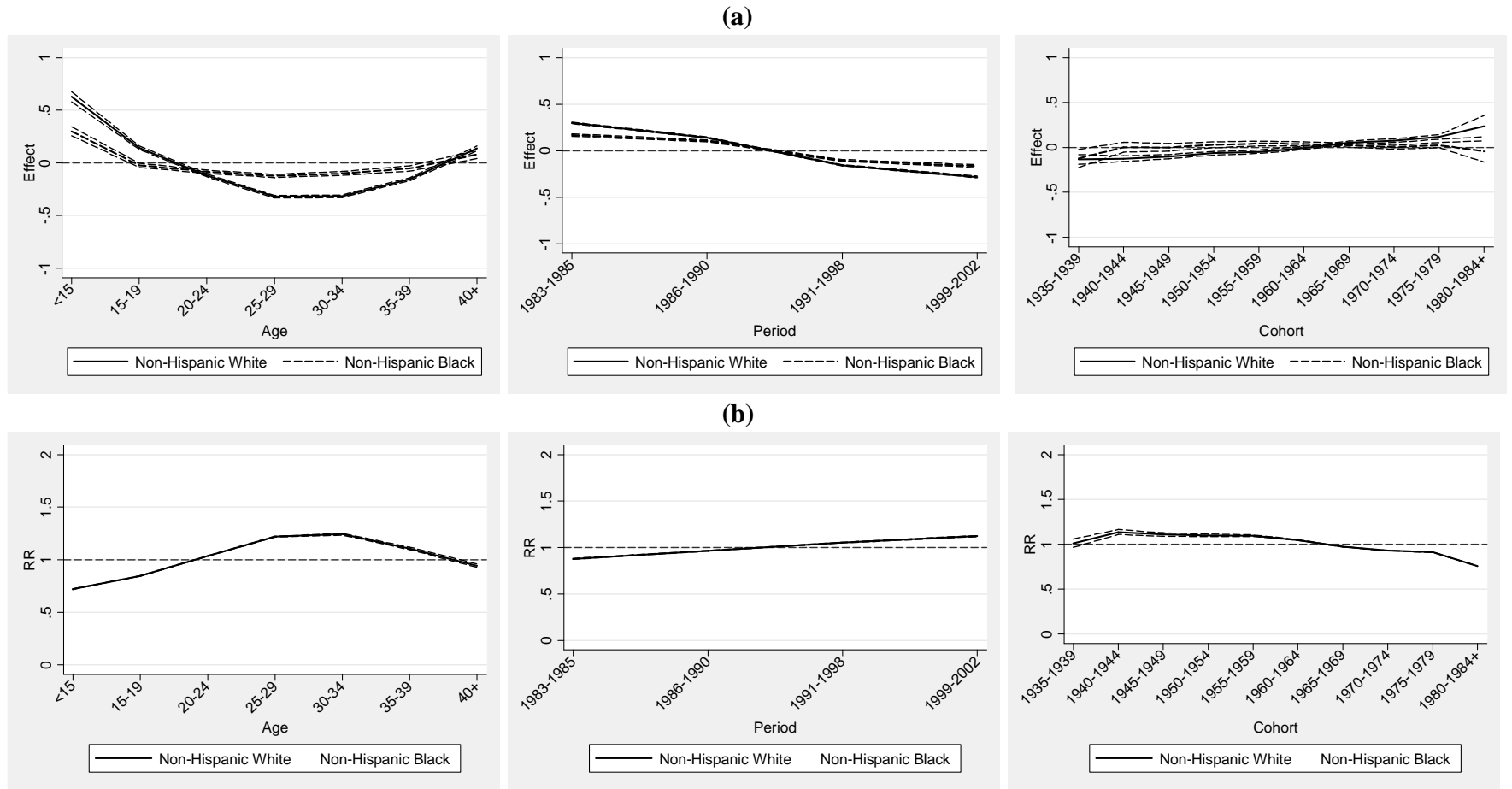


Figure 5 (a) Age, period and cohort estimates (log coefficients) and 95% confidence intervals. (b) Black/white age, period, and cohort relative risk ratios and 95% confidence intervals.