US infant mortality and the President’s party

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Background Infant mortality rates in the US exceed those in all other developed countries and in many less developed countries, suggesting political factors may contribute.

Methods Annual time series on overall, White and Black infant mortality rates in the US were analysed over the 1965–2010 time period to ascertain whether infant mortality rates varied across presidential administrations. Data were de-trended using cubic splines and analysed using both graphical and time series regression methods.

Results Across all nine presidential administrations, infant mortality rates were below trend when the President was a Democrat and above trend when the President was a Republican. This was true for overall, neonatal and postneonatal mortality. Regression estimates show that, relative to trend, Republican administrations were characterized by infant mortality rates that were, on average, 3% higher than Democratic administrations. In proportional terms, effect size is similar for US Whites and Blacks. US Black rates are more than twice as high as White, implying substantially larger absolute effects for Blacks.

Conclusions We found a robust, quantitatively important association between net of trend US infant mortality rates and the party affiliation of the president. There may be overlooked ways by which macro-dynamics of policy impact microdynamics of physiology, suggesting the political system is a component of the underlying mechanism generating health inequality in the USA.

Keywords Infant mortality, racial disparities, health inequality, political parties

Introduction

Much evidence shows that the US political realignment of the 1960s marked a critical, issue-based departing point between the Democratic and Republican parties.1 The Democratic Party distanced itself from the long-protected racially segregated ‘Jim Crow’ system in the South and reframed its policy platform around civil rights and the War on Poverty, whereas the Republican Party focused its platform on economic conservatism, employing the ‘Southern Strategy’ to capture votes from Democrats disaffected by the party’s civil rights platform.2,3 The frequency of cross-party coalitions in most key policy issues has diminished since the 1970s, and more radically during the past decade. Research has demonstrated that American politicians have become increasingly polarized along partisan lines.4

Given his many legislative, regulatory and budgetary powers, the President of the USA has a primary role in enacting his party platform while in office. Research has suggested, for example, that postwar macroeconomic policy in the USA reflects the economic interests of the party that controls the
presidency. Some studies of US income inequality lean toward political explanations as an alternative to technological and globalization paradigms. In an early example, a 1978 study indicated that partisan, short-term manipulations of economic policy, especially during electoral periods, could have longer-term social costs. More recently, a 2008 analysis argued that variations in economic growth linked to presidential partisan regimes have played a central role in the rise of income inequality in the USA since World War II. Given the connections established between the socioeconomic environment and health, and given the power of political parties to influence the policies that, for example, drive macroeconomics, it is reasonable to posit that political ideologies may affect health, either directly or indirectly.

Most empirical research to date in ‘political epidemiology’ has consisted of cross-national, often cross-sectional comparisons [predominantly among varying subsets of Organization for Economic Cooperation and Development (OECD) countries] considering questions related to associations between types of political regimes (e.g., welfare states or others; or among welfare states, democracies vs others, or social, Christian or liberal democracies compared with each other); expenditures on social policies; or level of income inequality and comparative population health (see Muntaner et al., and Beckfield and Krieger for reviews of the literature). For example, in a series of comparative studies on OECD nations, Navarro and colleagues identified that government type, party ideology and the generosity of welfare and labour market policies correlate with public health outcomes.

More recently, researchers have taken the approach of comparing changes in health over time across countries (see Lundberg et al., Granados, Regidor et al., and Avendano, with mixed conclusions. Taking this approach, Granados and Redigor et al. found a convergence in infant mortality rates across OECD countries, raising questions about the validity of cross-sectional findings. Avendano found that correlations between population health and income inequality were virtually eradicated when authors controlled for permanent differences across countries using country-specific fixed effects, compared with simple cross-country correlations, leading this author to question whether cross-country designs allow causal inference from the associations between specific welfare state characteristics and population health. However, Lundberg et al. analysed changes over time in 18 OECD countries and concluded that the design and generosity of family policies could measurably affect the infant mortality rate in the specific circumstance of supporting dual-earner families. Stuckler et al. found changes in social expenditures per person lowered all-cause mortality whereas changes in medical care expenditures did not, suggesting that the null findings of Granados, Redigor et al. and Avendano may, in part, reflect their choice of explanatory variables. This literature suggests that political factors can have causal effects on health outcomes, although a focus on income inequality or medical care expenditures across countries, per se, may miss the role that different political regimes may play in promoting population health under conditions of inequality, or in buffering the disadvantaged in unequal societies from the worst health consequences of inequality.

As a group, the reviewed studies suggest the importance of considering changes over time, of looking at specific policies or at political climates that generate policy, and of gaining additional leverage by examining the health impact of changes in politics or policies within a single country as an important complement to extant cross-country studies. Political actors, bureaucracies and institutions ultimately play critical roles in setting health and social policy agendas and implementing them. Additionally, politics qua ideology is central to framing such agendas and may vary across countries or be contested within countries.

In this line of reasoning, the divergent policy prescriptions of the political party holding sway at the US federal level may affect variation in national health indicators. Although the USA can be characterized as a liberal Democratic society, rather than treating the US as static in its politics, our approach acknowledges shifts in the balance of power between political traditions associated with the two major political parties within the USA. These shifts may proxy for or be the drivers of varying political climates or policy agendas that influence population health.

Researchers have reported within-country associations between the political party at the helm and temporal patterns of suicide rates, finding suicide rates are higher under more conservative regimes. In light of this evidence one can wonder whether the political forces that influence deaths due to an existential cause of death, such as suicide, would impact broader measures of population health or deaths due to other causes. We consider this possibility by asking the question: ‘Is the political party of the president of the USA associated with an important, objective and sensitive measure of population health, infant mortality?’

Methods

National and racial infant, neonatal and postneonatal mortality rate data are from the US National Vital Statistics Reports. All mortality rate measures are per 1000 live births. Since during the early part of the period we are considering—1965–2010, across nine presidents—it is not possible to distinguish non-Hispanic Whites and Blacks, for consistency we have included Hispanics in all of our tabulations. During the period when it is possible to distinguish the
non-Hispanics, doing so makes little difference to the reported rates.

Given that US infant mortality is strongly related to long-term factors (e.g. the advancement of medical technology, sanitation, Medicaid eligibility), all variables except the president’s party indicator were detrended. In the case of all mortality rates, the data were de-trended after being logged. A median cubic B-spline was fitted\textsuperscript{29–32} (Figure 1) and residuals were subsequently recovered; these residuals constituted the variables used in the analyses.

Cubic splines are a standard method for fitting smooth, non-linear functions to data. Cubic polynomials are fit between generally preset ‘knots’. Unlike linear splines, cubic splines are smooth around the ‘knots’. Because of the log transformation of mortality rates, our dependent variables (i.e. residualized logs of mortality rates) represent the natural logarithm of the ratio of annual observed mortality rates to the time-predicted annual mortality rate; explicitly, this ratio constitutes an estimate of the proportional rate of change of mortality rates net of variation assignable to historical trends. To avoid overfitting and to diminish autocorrelation between the knots, we used knots fixed at minimum and maximum values of time range plus equidistant internal knots fixed at 33.3% and 66.7% of time range.\textsuperscript{33} Altering the number of knots, or using alternative methods such as

Figure 1 Logged IMR, NMR, and PMR trends, 1965–2010
To formally test the association between presidential party affiliation and de-trended infant mortality rates, we estimated ordinary least squares (OLS) time series regressions. Standard errors have been calculated using the Newey-West estimator, which is robust to arbitrary forms of serial correlation. In the results we report, the error structure is assumed autocorrelated up to a maximum lag of 3 years, but results were robust to using maximum lags of 1, 2 or 4 years. It is well known that, in finite samples, the Newey-West estimator tends to underestimate sampling variability. We used recently developed fixed-bandwidth asymptotics to adjust the 95% confidence intervals and P-values. In our case, this adjustment had little impact on the estimated confidence intervals or P-values.

Obviously, many factors other than presidential party affiliation affect infant mortality, including social factors that presidents may have little power to influence. Many such factors should move slowly over time. As a result, flexibly de-trending the data should have effectively controlled for the potential confounding effect of these factors. In addition, we experimented with controlling for a variety of other factors that have been found to affect infant mortality rates. (See Table 1 for definitions of study variables.) In our case, we found that results on president’s party are robust to such model specifications.

**Results**

As Figure 1 indicates, over the past 46 years infant mortality rates in the USA have declined dramatically, dropping 75% from 24.7 in 1965 to 6.1 in 2010, with this trend representing the dominant feature of the data. The infant mortality rates for Blacks remain more than twice what they are for Whites. However, if one removes the trend, one sees a pattern with overall and race-specific infant mortality rates being close to 0.02 ln points (2%) above trend during a typical Republican administration year and just over 0.01 ln points (1%) below trend during a typical Democratic administration year. This pattern is even more evident if one plots the residuals from trend as we do in Figure 2. There we see the pattern exists not only for infant mortality rates but also when we break these rates into neonatal and postneonatal mortality. Table 2 presents our results in the context of time series regressions, where all variables, with the exception of the presidential party indicator, have been de-trended. Because the explanatory variables we use are available over different intervals, we first report

### Table 1 Descriptive statistics of de-trended variables

<table>
<thead>
<tr>
<th>Definition</th>
<th>Variable (de-trended)</th>
<th>Mean Dem</th>
<th>Mean Rep</th>
<th>Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent of individuals under the federal poverty level</td>
<td>Poverty rate (All races)</td>
<td>-.16</td>
<td>.10</td>
<td>1965–2010</td>
</tr>
<tr>
<td></td>
<td>Poverty rate (Whites)</td>
<td>-.11</td>
<td>.07</td>
<td>1965–2010</td>
</tr>
<tr>
<td></td>
<td>Poverty rate (Blacks)</td>
<td>-.69</td>
<td>.42</td>
<td>1966–2010</td>
</tr>
<tr>
<td>Percent of non-institutionalized persons 25 years or over who completed 4 years of high school education or more</td>
<td>Education (All races)</td>
<td>.08</td>
<td>-.05</td>
<td>1965–2010</td>
</tr>
<tr>
<td></td>
<td>Education (Whites)</td>
<td>.08</td>
<td>-.05</td>
<td>1965–2010</td>
</tr>
<tr>
<td></td>
<td>Education (Blacks)</td>
<td>.22</td>
<td>-.14</td>
<td>1965–2010</td>
</tr>
<tr>
<td>Unemployed as percent of the civilian labour force (16 years of age and over)</td>
<td>Unemployment rate (All races)</td>
<td>-.22</td>
<td>.14</td>
<td>1965–2010</td>
</tr>
<tr>
<td>Income share of individuals at top 5% in the family income distribution</td>
<td>Income share top 5% (All races)</td>
<td>.06</td>
<td>-.04</td>
<td>1965–2010</td>
</tr>
<tr>
<td>Income share of individuals at bottom 20% in the family income distribution</td>
<td>Income share bottom 20% (All races)</td>
<td>.05</td>
<td>-.03</td>
<td>1965–2010</td>
</tr>
<tr>
<td>Adult (17+ years) per capita yearly consumption of US manufactured cigarettes</td>
<td>Smoking (All races)</td>
<td>7.98</td>
<td>-5.42</td>
<td>1965–2006</td>
</tr>
</tbody>
</table>

bivariate specifications for the 1965–2010 period and then for the 1966–2010 period. Specification 3 includes income, education and unemployment rate controls, whereas specification 4 adds smoking to this. In no case does the inclusion of these variables have a significant effect on the estimated coefficient on the presidential party indicator. Essentially, the regression results replicate what we see in the figures—relative to trend, infant mortality rates are, on average, 3% higher during Republican administrations. With the exception of the results on postneonatal mortality for Blacks, the estimates are quite precisely estimated. In proportional terms, effects on overall infant mortality are very similar for Blacks and Whites. The estimates for neonatal mortality suggest somewhat stronger effects for Blacks than Whites, though this difference is imprecisely estimated (Δβ = .013, ± .020 for model 1 and Δβ = .012, ± .020 for model 2). Alternatively, the estimated proportional effect on postneonatal mortality is higher for Whites than for Blacks although, once again, this difference is not very precisely estimated (Δβ = −.014, ± .015 for model 1 and Δβ = −.017, ± .015 for model 2).

**Discussion**

We have described a quantitatively important, robust association between net-of-trend US infant mortality rates and the party affiliation of the president. Relative to trend, national and race-specific infant, neonatal and postneonatal mortality rates decrease under Democratic administrations and increase under Republican administrations.
Table 2 Parameter estimates of the effect of presidential party (Republican) on infant mortality

<table>
<thead>
<tr>
<th></th>
<th>Ln infant mortality</th>
<th></th>
<th>Ln neonatal mortality</th>
<th></th>
<th>Ln postneonatal mortality</th>
<th></th>
<th></th>
<th></th>
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<tr>
<td></td>
<td>All races</td>
<td>Whites</td>
<td>Blacks</td>
<td>All races</td>
<td>Whites</td>
<td>Blacks</td>
<td>All races</td>
<td>Whites</td>
<td>Blacks</td>
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<td></td>
<td>Coefficient estimate</td>
<td></td>
<td></td>
<td>Coefficient estimate</td>
<td></td>
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<td>Coefficient estimate</td>
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<tr>
<td>(1)</td>
<td>.031</td>
<td>.029</td>
<td>.031</td>
<td>.024</td>
<td>.019</td>
<td>.032</td>
<td>.038</td>
<td>.043</td>
<td>.029</td>
</tr>
<tr>
<td></td>
<td>95% confidence interval</td>
<td></td>
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<td>95% confidence interval</td>
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<td>95% confidence interval</td>
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<td></td>
<td>±.016</td>
<td>±.013</td>
<td>±.022</td>
<td>±.012</td>
<td>±.011</td>
<td>±.020</td>
<td>±.029</td>
<td>±.027</td>
<td>±.032</td>
</tr>
<tr>
<td></td>
<td>P-value</td>
<td></td>
<td></td>
<td>P-value</td>
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<td>P-value</td>
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<tr>
<td></td>
<td>.000</td>
<td>.000</td>
<td>.007</td>
<td>.000</td>
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<td>.003</td>
<td>.013</td>
<td>.002</td>
<td>.076</td>
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<tr>
<td>(2)</td>
<td>Coefficient estimate</td>
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<td>.030</td>
<td>.031</td>
<td>.026</td>
<td>.020</td>
<td>.030</td>
<td>.044</td>
<td>.027</td>
</tr>
<tr>
<td></td>
<td>95% confidence interval</td>
<td>±.016</td>
<td>±.013</td>
<td>±.023</td>
<td>±.011</td>
<td>±.011</td>
<td>±.030</td>
<td>±.027</td>
<td>±.034</td>
</tr>
<tr>
<td></td>
<td>P-value</td>
<td>.000</td>
<td>.000</td>
<td>.010</td>
<td>.000</td>
<td>.001</td>
<td>.013</td>
<td>.002</td>
<td>.106</td>
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<tr>
<td>(3)</td>
<td>Coefficient estimate</td>
<td>.034</td>
<td>.031</td>
<td>.031</td>
<td>.027</td>
<td>.022</td>
<td>.044</td>
<td>.046</td>
<td>.022</td>
</tr>
<tr>
<td></td>
<td>95% confidence interval</td>
<td>±.011</td>
<td>±.008</td>
<td>±.024</td>
<td>±.009</td>
<td>±.013</td>
<td>±.024</td>
<td>±.024</td>
<td>±.036</td>
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<tr>
<td></td>
<td>P-value</td>
<td>.000</td>
<td>.000</td>
<td>.013</td>
<td>.000</td>
<td>.002</td>
<td>.001</td>
<td>.000</td>
<td>.218</td>
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<tr>
<td>(4)</td>
<td>Coefficient estimate</td>
<td>.033</td>
<td>.031</td>
<td>.029</td>
<td>.030</td>
<td>.027</td>
<td>.035</td>
<td>.033</td>
<td>.016</td>
</tr>
<tr>
<td></td>
<td>95% confidence interval</td>
<td>±.013</td>
<td>±.010</td>
<td>±.026</td>
<td>±.009</td>
<td>±.011</td>
<td>±.027</td>
<td>±.024</td>
<td>±.034</td>
</tr>
<tr>
<td></td>
<td>P-value</td>
<td>.000</td>
<td>.000</td>
<td>.031</td>
<td>.000</td>
<td>.000</td>
<td>.014</td>
<td>.010</td>
<td>.349</td>
</tr>
</tbody>
</table>

The error structure is assumed heteroskedastic and autocorrelated up to a maximum lag length of three years. The P-values and 95% confidence intervals are adjusted using fixed-bandwidth asymptotics. All coefficients are for Republican presidents (Republican = 1, Democratic = 0). Model 1 is without controls for data between 1965 and 2010 (n = 46). Model 2 is without controls for data between 1966 and 2010 (n = 45). Model 3 controls for unemployment rate, poverty rate, education, income share of top 5%, and income share of bottom 20%, 1966–2010 (n = 45). Model 4 adds smoking to Model 3, 1966–2006 (n = 41). These confounders were used as national indicators for ‘All races’ models, and race-specific indicators (with the exception of unemployment rate, income shares, and smoking) for ‘Whites’ and ‘Blacks’ models (e.g. if the dependent variable was Ln White infant mortality rate, the model controls for poverty rate for Whites and education for Whites).
Infant mortality rates are about 3% higher during a typical Republican-president year compared with a typical Democratic-president year. In proportional terms, effects are roughly as large for Blacks as they are for Whites. However, since Black rates are more than twice as high as White rates, this implies substantially larger absolute effects for Blacks.

To put the magnitude of these findings in context, Currie and Gruber\(^{38}\) report that policy changes between 1979 and 1992 that expanded the eligibility for Medicaid—a joint federal-state health insurance entitlement programme for the poor—by roughly 30% induced an 8.5% decline in infant mortality. Thus, the magnitude of the effects we estimated are about one-third of the magnitude of the effect that Currie and Gruber attributed to the substantial Medicaid expansions over the 1980s. We note the timing of this Medicaid expansion does not fit the timing of the patterns we observe, suggesting other factors explain the association between the president’s party and infant mortality.

This is the first study to our knowledge to focus within a country, over a substantial time period, on changes in an explicitly political variable, presidential party, and its association with a sensitive measure of population health, infant mortality. The importance of the president’s party as a correlate of infant mortality is accentuated when considered in light of the relatively slow pace of significant changes in the social determinants of health (e.g. a significant change in the national average level of education) compared with the 4- or 8-year cycles of presidential party administrations.

Perhaps it is not surprising to see national-level effects of the Great Society policies introduced by President Johnson during the 1960s. Collectively, the advancements of the Civil Rights Movement, the 1964 Civil Rights Act, the Voting Rights Act of 1965, the Higher Education Act of 1965, the Social Security Act of 1965 and the War on Poverty had fundamental effects on life within the USA, and empirical studies\(^{39–41}\) suggest a lasting legacy of the policies introduced during the Johnson administration on health outcomes and health disparities. However, we have also found a substantial presidential party effect since then.

What might explain this association? Perhaps the association is spurious, in the sense that there are autonomous factors shifting back and forth with a period of about 4 or 8 years. Without knowing the mechanisms connecting presidential party affiliation and infant mortality, we cannot completely dismiss this possibility, but are striking the consistency of the association we have uncovered. If the association is not spurious, it could arise because of selective fertility: perhaps high-risk infants are more likely to be born during Republican administrations. For example, there is evidence that aborted fetuses would have been more likely to die as infants than those carried to term.\(^{42–44}\) We conducted a crude test for overall selection into motherhood by including de-trended birth rate (BR) and abortion rate (AR) variables in our estimating equations. The coefficient on the presidential party indicator was robust to the inclusion of ARs and BRs in our models. (See Supplementary Table 2 available as Supplementary data at IJE online.)

Alternatively, the association could arise because of conditions existing for mothers and infants during Democratic vs Republican administrations. Findings from the social epidemiological literature show that the distribution of health outcomes is highly responsive to life experiences and resources.\(^{45}\) The processes through which the different systems of the human body adapt to daily life challenges have considerable effects on physiological indicators of health risks.\(^{46–48}\)

To the extent that the respective traditions of the two US political parties promote the interests or well-being of different US constituencies, or view health disparities through different political ideological lenses, the party in power may influence infant mortality rates. For example, it may be that Republicans are more likely to view health disparities as inevitable, whether due to market forces or as a matter of personal responsibility to be addressed through individual health behaviors, not by government. Democrats, on the other hand, may be more likely to view health disparities as a preventable social problem about which something should be done via government intervention. Although this is an oversimplified rendering of the differences in the ideological traditions, it suggests one type of reason why one might expect Republican and Democratic presidents to affect population health differently. For example, whether a government advocates austerity measures or increasing social welfare protections as the response to economic crisis may reflect such ideological differences and influence mortality rates.\(^{20,49}\)

To the extent that increasingly polarized Republican and Democrat politicians represent coalitions with constituencies that differentially suffer the costs or enjoy the benefits of social policies that influence population health, this might also affect their respective priorities towards promoting population health. Similarly, to the extent that the election of the president from one party or the other reflects the empowerment, enfranchisement or political mobilization of—vs the suppression of—different groups, the health of vulnerable populations may be impeded or promoted as either cause or effect.\(^{50}\)

This is not to say that the president’s party is the only or most important political factor influencing US population health. The USA is a large and diverse country, with three branches of federal government and subnational levels of government. Presidential effectiveness in promoting specific agendas may vary with the party compositions of other federal branches or at state levels. For example, recent polarization...
between Republicans and Democrats in Congress on the extent to which the Agriculture Bill should include farm subsidies or support for the Supplemental Nutritional Assistance Program (food stamps) led to the first Agriculture Bill since 1973 that did not include food stamps, a partisan position at odds with that of the current President’s party. The 2012 decision by a right-leaning Supreme Court was to uphold the individual mandate that is central to Democratic President Obama’s signature health legislation, the Affordable Care Act (ACA), while allowing states to opt out of the law’s Medicaid expansion. Several Republican governors are refusing to participate in the Medicaid expansion. However, the response among Republican governors has not been uniform; other initial opponents of the ACA are choosing to participate in the expansions now that they are a matter of law, indicating that a president from one party can influence the party discipline of opposite-party state executives.

The current paper adds to the extant record on infant mortality and disparities in infant mortality rates by demonstrating a powerful, robust association between variations in US infant mortality rates over the past half-century and the inclusion of a political variable: the political party of the president. Further research is needed to determine whether the association we have uncovered is causal, and to identify the mechanisms involved.

**Supplementary Data**

Supplementary data are available at *IJE* online.

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**Conflict of interest**: None declared.

**KEY MESSAGES**

- Since 1965, net-of-trend US infant mortality rates (IMR) are associated with the party that holds the presidency, with IMR decreasing under Democratic administrations and increasing under Republican Administrations.
- This 3% gap holds for neonatal and postneonatal mortality rates and for US Blacks and Whites, implying larger absolute effects for Blacks.
- Findings are robust to controls for income, education, unemployment, smoking and birth and abortion rates, as well as to statistical de-trending methods.
- Further research is needed to determine whether the association is causal, and to identify mechanisms.

**References**
