ORIGINAL RESEARCH ARTICLE

Elections have Consequences: Partisan Politics may be Literally Killing Us

Alexander Maas[1] · Liang Lu[1]

Abstract

Background Presidential campaigns and election outcomes have significant health implications for voters and communities. The theoretical underpinning of this relationship is multifaceted, but a new and growing field of empirical literature strongly suggests communities that voted for the losing presidential candidate may experience decreased physical and mental health under the leadership of the winning candidate.

Objective Our objective was to estimate the relationship between mortality rates and community support for the losing presidential candidate (partisan loss).

Methods Mortality data compiled by the US Centers for Disease Control and election results at the county level were used across a suite of county-year fixed-effects models to estimate the effect of election outcomes on mortality rates for the years 1999–2017.

Results Mortality rates were positively associated with partisan loss. Results suggest mortality rates increase by as much as 3% in extremely partisan counties following presidential election losses.

Conclusions We suggest two mechanisms—social disintegration and/or partisan theory—by which mortality rates are likely to increase for counties that voted for the losing presidential candidate.

Key Points for Decision Makers

Counties that vote strongly for the losing presidential candidate appear to experience increased mortality rates in subsequent years.

Increase mortality rates appear strongest in the second and third year after an election.

The winner-take-all system used in the two-party presidential election system may have significant unacknowledged costs for supporters of the losing candidate.

✉ Alexander Maas
  alexmaas@uidaho.edu

1 Department of Agricultural Economics and Rural Sociology, University of Idaho, 875 Perimeter Dr, Moscow, ID 83844, USA

1 Introduction

Presidential candidates in the USA spend the better part of a year energizing potential voters and dissuading constituents from supporting other candidates. While campaigns have different tones and themes—some more unifying than others—a recent Pew Report found that the polarization of American politics and partisan antipathy are at a modern-day high [1, 2]. Many researchers have begun to worry that hyper-partisanship will have detrimental effects on our national identity and dialog [3–6] since presidential candidates often espouse starkly different beliefs and ideologies [7, 8] that encourage some voters and worry others. This paper investigates whether such worries are justified by answering the following question: Do communities that voted for the losing candidate experience measurable differences in mortality rates?

Much of the research to date linking politics to health has been dictated by party comparisons. For example, Rodriguez et al. [9] found that Republican administrations were associated with an increase in infant mortality rates of approximately 3% compared with Democratic administrations. Similarly, suicide and homicide rates appear to increase under

conservative government regimes, although the causality of this relationship remains unclear [10–12]. However, given the growing partisan divide in recent decades [13, 14], a growing body of literature has begun investigating not only the effect of a political party but also the effect that voting for a losing presidential candidate—referred to as partisan loss (PL) [15]—has on health outcomes. While research into the social determinants of health is extensive [16, 17], there is a dearth of empirical work investigating the magnitude and mechanisms by which PL impacts health. To date, no research has empirically estimated the effect of PL on mortality rates specifically. Although our work is unable to quantify the effects of specific mechanisms by which PL influences mortality, we present two complementary explanations proposed in previous work.

The first explanation is psychosocial, where increased feelings of isolation or elevated anxiety that accompany supporting the losing candidate are correlated—and to some extent cause—poor health and potentially acute causes of death—cardiovascular events or suicide, for example [18, 19]. This explanation is largely based in social cohesion theory, which suggests that individuals' social connection to their community is critical in promoting health and wellbeing [20]. Under this paradigm, the election of a president whose views differ from one's own serves as a shock to social cohesion.

The second explanation is institutional: represented by the basic tenets of partisan theory, which suggests that voters rationally choose politicians whose policies provide them socioeconomic benefits [21–23]. Thus, politicians from each party attempt to enact divergent policies that benefit their supporters, potentially at the expense of those who oppose them. It is reasonable to think such preferential policy design can result in poorer health outcomes for communities that oppose the acting administration (see discussion in Wigley and Akkoyunlu-Wigley [24]). In this way, one cause of increased mortality is psychosocial (perspectives and feelings of belonging with a community) and the other is socioeconomic, a direct result of policies that impact on household economic conditions heterogeneously and thereby health and mortality.

Separately identifying the effect of each phenomenon is not feasible. As such, this analysis is empirical in nature and attempts to identify the overall effect of PL with neutrality toward the underlying mechanisms.

We focus on the presidency because this office is likely to exert the strongest institutional and psychosocial effect. The President of the USA has a disproportionate ability to enact their party's platform while in office—particularly when other branches of government are deadlocked [25]. As such, the Executive Branch of the USA has significant social, legal, and political power, that may have distributional impacts across constituents. Generally, presidents

have two options for pursuing policy: working with congress to craft bills into laws or by exercising unilateral powers as executive orders of agency guidelines, thereby creating policies with the weight of law but without formal endorsements [26]. Thus, the president has significant legislative power to affect the citizenry that is unmoderated by compromises that may occur in the legislative branch.

Accordingly, this paper uses presidential election outcomes to empirically investigate the short-term impact of PL on mortality rates. While two possible mechanisms for such relationships are proposed, the current investigation does not separately identify their effects.

2 Literature Review

2.1 Anxiety, Stress, and Isolation

The public health model suggests that environmental factors (economic, social, and political) play a role in health outcomes, although the mechanisms and significance of this relationship are a topic of considerable debate [27–30]. While many factors affect human health and wellbeing, researchers generally agree that anxiety and social disintegration are correlated with poorer outcomes. The decline in America's health and increased mortality rates are issues that have been raised in the public health literature since the 1990s and speculatively linked to worsening psychosocial factors [31]. For example, growing evidence suggests a strong relationship between psychosocial factors (e.g., depression or chronic stress) and cardiovascular disease, particularly among those of low socioeconomic status [32, 33]. The election of a president who is antithetical to one's own identity or local community may have significant impacts on social cohesion and trust, which in turn has strong implications for health and mortality [20, 34, 35].

Recent work examining welfare indicators as a function of partisan electoral losses suggested that constituents who lose at the ballot box experience a quick and significant decrease in happiness after an election. Such losses have a stronger effect on public wellbeing than the Newtown shootings or the Boston Marathon bombing [15]. Motyl et al. [36] found that individuals who voted for the losing candidate experienced decreased feelings of belonging and increased thoughts of migration. In contrast, individuals who supported the current government tended to think more optimistically about their economic future [37], which may reduce anxiety, since optimism has been linked with working harder, later retirement, marriage, and increased savings [38]. Given the well-documented phenomenon of partisan attachment—members of a political party are more likely to trust and share resources with other members of that party [39, 40]—these results are not surprising.

Elections not only affect subjective measures of happiness but have also been associated with physiological changes. Waismel-Manor et al. [41] found changes in self-reported Positive and Negative Affect Schedule that corresponded with elevated cortisol levels—a hormone linked to stress— among the voting public on election nights. Following elections, voters supporting the losing candidate experienced marked drops in testosterone levels and increases in cortisol [42, 43]. After the 2008 elections (where Democratic candidate Barack Obama was elected), individuals who identified as conservative were more negative in emotional responses to surveys and experienced a spike in salivary cortisol levels [43].

Research linking election outcomes and health is limited, but a growing field of literature suggests presidential campaigns (and other election outcomes) may have significant health implications for voters and communities. Winning candidates that support and express the ideas of historically disenfranchised groups have been associated with short-term health improvements for those communities [44]. By contrast, the election of officials who are hostile to social-political groups has been linked to increased stress levels in those groups. In a perspectives piece, Morey [45] discussed the mechanism by which anti-immigration politics can detrimentally affect the health of immigrant and Hispanic communities, highlighting that many of these mechanisms may be social, increasing anxiety regardless of explicit changes to policy or healthcare access.

Indeed, some data support Morey's implicit hypotheses: US states with a more exclusionary immigration policy experience higher rates of poor mental health among Latinos [46], although endogeneity concerns limit the causal inference of these results. Following the 2016 election, where Republican candidate Donald Trump was elected, there was a significant uptick in preterm deliveries by Latina women [47]. While the relationship is correlative, depression and anxiety have also been linked to spontaneous preterm births among African-American women [48]. Conversely, work by Classen and Dunn [49] suggested that suicides may decrease with PL, since such losses may improve social integration among communities.

Although empirical research is limited, the current consensus in the literature suggests that PL, or living in politically incongruent environments, can induce anxiety, distrust, and feelings of isolation [39, 50–52]. These findings suggest the potential for observable negative health outcomes, particularly for people who believe their communities are targets of hostility or discrimination [53]. Thus, even without policy shifts, the anxiety or social factors caused by election losses may directly affect health. Of course, empirical evidence does suggest partisan cycles and preferential partisan policies exist, which have distributional effects on human

welfare if politicians enact policies that disproportionately benefit their supporters [54, 55].

2.2 Partisan and Preferential Policies

The extent that worry over voting for the losing candidate is justified largely depends on the polity of presidential leadership and the policies they create (or reverse). Theoretical and empirical work evaluating electoral-business-cycle interactions and outcomes has found mixed results but generally suggests that incumbents may manipulate micro- and macroeconomic conditions to benefit themselves electorally [56–58] or reward their supporters [59–61]. The extent to which this manipulation is possible depends on (1) international and domestic, (2) political-economic, and (3) institutional and structural contexts [62].

Although the degree of manipulation may vary across contexts, partisan theory suggests that, as part of their electoral-seeking behavior, competing political parties cultivate relationships with different voting blocs by nurturing reputations for policy making that favor those groups. As a result, counties that favored the winning candidate will disproportionally benefit under their leadership.[1] Indeed, recent work by Reingewertz and Baskaran [23] suggested that presidents provide more federal outlays to districts represented by their co-partisans.

Some of these policies directly affect socio-demographic groups, whereas others influence macroeconomic conditions that indirectly favor particular constituents. For example, social policies, such as the legalization of same-sex marriage or the expansion of Medicare, have specific targeted populations. By comparison, policies that affect interest rates or income taxes may not target specific beneficiaries but affect macroeconomic conditions that have distributional impacts across partisan and socioeconomic groups. For the majority of the twentieth century (although somewhat different in recent history), a clear distinction of left- and right-party policy was the relative importance placed on inflation or unemployment. Leftist parties accept higher inflation to obtain lower unemployment and higher growth; rightist parties tolerate higher unemployment and lower growth to obtain lower inflation [62]. These policy trade-offs have implications for socioeconomic groups, where those at the low end of occupational and income hierarchies are disproportionately hurt by unemployment and recessions that are only partially offset by tax-and-transfer systems [63].

---

[1] Note that we omit a common discussion of the median-voter theory [64] and partisan theory [65, 66] for brevity and accept the "overwhelming empirical evidence [that] shows legislators regularly take positions that diverge significantly from the preferences of the median voter in their districts" [67].

Health policies enacted by presidential administrations also have direct distributional impacts. For example, the Affordable Care Act (ACA) significantly increased health insurance enrollment [68]. Barbaresco et al. [69] found that, among young adults, the ACA increased the probability of having health insurance, a primary care doctor, excellent self-assessed health, and a reduction in body mass index. Over the last 50 years, the use of federal funds to sponsor Community Health Centers reduced mortality by 2% among individuals aged > 50 years [70], whereas Medicare led to a sharp increase in medical services for those aged ≥ 65 years [71]. At the state level, policies involving tobacco, labor, immigration, civil rights, and the environment have also been linked to life expectancy, where policies in bluer states result in a > 2-year increase in life expectancy [72], although the direction of causality in such findings is questionable. Recently, the heterogeneous responses across states to the COVID-19 pandemic is clear evidence that policies enacted by executive branches may have disproportional health impacts across communities (particularly communities of color) [73, 74].

Economic and health policies have obvious implications for health outcomes and mortality [75, 76], but agricultural, environmental, infrastructure, or defense policies may also have distributional health impacts across locations and socioeconomic groups [77–81]. As such, many policies enacted under partisan administrations may affect mortality, if only indirectly.

While policy has obvious implications for affecting distributional health outcomes, it is difficult to separately identify the institutional effect of policy from the psychosocial effect constituents experience as a result of that policy and the politicians who enacted it. As such, we reiterate that the following analysis investigates the link between presidential election outcomes and mortality rates of communities who voted for or against that president, but it does not make claims about the mechanisms underlying this relationship. Indeed, it is likely that both mechanisms operate simultaneously and cannot be separately identified without (infeasible) experimentation.

3 Data and Estimation

Estimating the effect of a particular leader or election on the economy or constituents is complicated by potential reverse causality. Simply put, voters' expectations about the future (whether accurate or not) may affect both who wins the election and how future policies manifest. This concern has been thoroughly voiced in the literature, particularly with respect to interest rates and unemployment [62]. While these concerns are valid in many analyses, using presidential election results as an explanatory variable in county-level mortality

rates helps ameliorate concerns of endogeneity for two reasons. First, individual counties have little effect on the election results, such that the eventual winning candidate is largely exogenous to the voting behavior of partisan counties. For example, in Bronx County, NY, approximately 90% of votes cast in presidential elections are for Democratic candidates. In elections where Democrats win, PL would be very low; in elections where a Republican wins, the loss score would be large, but in either case Bronx voters have little influence on the final electoral outcome.

Second, at the national level (and in swing counties), Republican and Democratic candidates share about 50% of the vote, such that the ultimate winner is largely uncertain and independent of particular counties. Thus, counties experience quasi-experimental shocks to PL whenever elections are essentially a coin toss. In fact, two of the five presidential elections in our sample had unexpected results,[2] where the candidate who won the popular vote ultimately lost the election. These unexpected election outcomes lessen endogeneity concerns over spurious correlations between economic or political expectations (in finance literature, similar use of surprise elections as event study "shocks" is common [82–84]). For example, if the expectation that a candidate would ultimately lose led to unhealthier behavior from their supporters today, our estimates of PL's effect on mortality would be biased, since the expectation of an election may influence mortality before its result.

3.1 Data

To test our hypothesis, that voting for a losing candidate increases (total, cardiovascular, and self-harm) mortality rates, a suite of models were estimated using age-adjusted crude rates (occurrence per 100,000) for males and females, aged 20–69 years, in each county, between the years 1999 and 2017. As is common in the literature, we ran separate models for men and women because they have significantly different mortality schedules, particularly for suicide and cardiovascular causes of death (COD). Our primary focus is on all-cause mortality rates, but more acute CODs such as cardiovascular and suicide are estimated individually because these conditions are more sensitive to acute changes in economic conditions, relative to long-term illnesses such as cancer [85]. The years included in our study are coded with the tenth revision of the *International Classification of Diseases* (ICD-10), which began in 1999. Comparability between the ICD-10 and the ninth revision (ICD-9) complicates classification since the National Vital Statistics System

---

[2] Beyond winning the popular vote, Al Gore and Hillary Clinton were both favored based on the odds given by common gambling websites and some national polls.

acknowledges a substantial discontinuity in trend for some CODs. Thus, we limit the analysis to years with ICD-10 coding. This temporal range provides sufficient variation as it includes the results from six elections and both Democratic and Republican presidencies. Crude rates were calculated using compressed mortality data provided by the National Center for Health Statistics of the Centers for Disease Control (CDC).

Voting data include county-level election results compiled by David Leip in the *Atlas of U.S. Presidential Elections*, which are used to create the three explanatory variables. These variables include a continuous (PL_c) and a discrete (PL_d) PL metric, as well as a dummy variable to indicate extremely partisan counties (EP). PL_c is calculated as the ratio of votes for the losing candidate over the sum of total votes for a major party candidate.[3] For example, if a Democrat won the election, $PL_c = \frac{\#\text{Votes For Republican}}{\#\text{of Votes For Republican}+\#\text{Votes For Democrat}}$ for each year in that election cycle. The discrete variable, PL_d, is calculated as 1 if more than 50% of the votes cast for a major party candidate were for the losing candidate and 0 otherwise. Extreme partisanship is calculated as 1 if more than 65% or less than 35% of the votes cast for a major party candidate were for the losing candidate and a 0 otherwise.

A complication in estimating models with an annual time step is the timing of elections. In election years, calculating the PL term is problematic because 10 months exist before, and 2 months exist after, the election. Moreover, there is conflicting evidence that ceremonial events, such as elections, exert their own effect on mortality rates [86–89]. To avoid weighting and other complications, election years were dropped from the sample when Equations (1–2) are estimated. PL_c is calculated as a continuous variable between 0 and 1, which represents the proportion of individuals in a county that vote for the losing candidate. This variable therefore remains constant across the 4-year term of the elected president. As such, each county is assigned six unique PL values to reflect the relative PL experienced by the county for a given election cycle each year in the sample.

In combining and cleaning these datasets, we omitted several counties and one state from the analysis. Alaska's boroughs could not be mapped consistently across voting and CDC datasets. Several additional counties were omitted if Federal Information Processing Standard boundaries or codes changed across the years in our sample. Despite these omissions, the dataset remained large, with 3026 distinct counties across 49 states and 19 years.

3.2 Models

Mortality rates (separated by COD) were included as dependent variables across a suite of regressions: all-cause mortality, suicide, and cardiovascular. Because mortality rates are necessarily positive (or zero) and positively skewed with a long right tail, each of the following equations is estimated using crude rate (y_{it}) and its $\sinh^{-1}(y_{it})$ transformation as the dependent variable. Fixed effects are used to control for unobserved differences across county and year, which should absorb any annual or geographic differences. Thus, the simplest model can be written as Eq. (1):

$$y_{it} = \alpha_i + b_t + \beta PL_{it} + \varepsilon_{it}, \tag{1}$$

where y_{it} is the mortality rate (total, suicide, or cardiovascular), α and b are fixed-effects terms for county and year, respectively, β is the coefficient of interest, and ϵ is a stochastic error term. Counties are indexed by i, and years are indexed by t. While this model specification allows for intercept differences in year and county, it may be too restrictive if factors influencing mortality vary across states over time. In this case, the model must allow for interactions of year and state factor variables and can be written as in Eq. (2):

$$y_{it} = \alpha_i + \sum_{j=1}^{49} \sum_{t=1}^{14} \rho_{jt} s_j b_t + \beta PL_{it} + \varepsilon_{it}, \tag{2}$$

where s_j is a set of dummy variables for each state and ρ are interaction-specific coefficients to be estimated. Equation (2) is preferred because it uses over 3600 dummy variables to control for geographic and temporal variation. However, Equation (2) may also over-control by absorbing potential PL effects in the state–year effect. For example, if a partisan policy affects the entire state, much of this effect will be absorbed in the fixed-effect term, instead of the PL term. Equations (1 and 2) also assume a linear and constant marginal effect of PL_c, which may be unrealistic in the presence of thresholds or non-linearities across election cycles or PL and do not allow for differing effects based on the temporal proximity to the election. It is reasonable to think that the effect of PL will vary across time, although the direction is unclear. Creating and implementing policy takes time, which suggests larger effects may exist later in the cycle, but the acute shock of loss may also diminish the further one gets from an election.

To capture these dynamics, Eq. (3) investigates the temporal relationship of PL and mortality within an election cycle. An interaction term is created by multiplying $PL_{d,it}$

---

[3] Note that lumping left and right parties together did not affect our results, but this version is not presented in our results as the policies of third-party candidates did not consistently align for or against the policies of the presidents' party. Moreover, logic dictates that voters supporting third-party candidates have no expectation of winning.
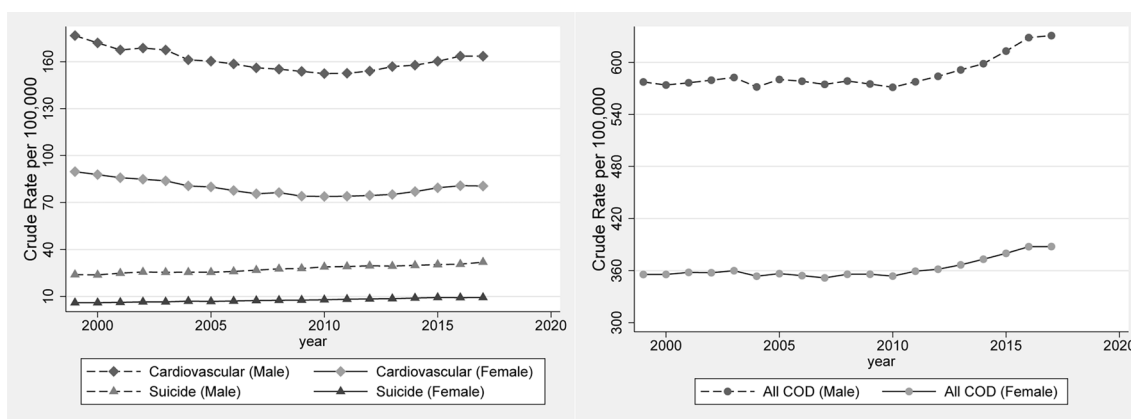
Fig. 1 Annual mortality trends of all cause of death, suicide, and cardiovascular death for males and females aged 20–69 years. *COD* cause of death

with a set of dummy variables representing years since an election, such that the model can be written as in Eq. (3):

$$y_{it} = \alpha_i + \sum_{j=1}^{49} \sum_{t=1}^{14} \rho_{jt} s_j b_t + \sum_{c=0}^{3} \vartheta_c v_c \text{PL}_{d,it} + \varepsilon_{it}, \qquad (3)$$

where v_c are dummy variables for each year after an election, indexed by $c \in [0, 3]$, and ϑ_c is the coefficient of interest. Equations (1–3) are estimated using population-weighted least squares, as is common when observational units have substantially different populations [90–92], and hypothesis testing is done using standard errors clustered on the county election cycle.

While model 1–3 specifications are reasonable, it is difficult to ex-ante determine the functional relationship of PL to mortality. As such, additional models with various underlying assumptions are included in the electronic supplementary material (ESM), each assuming a different structural relationship between PL and mortality.

3.3 Summary Statistics

A preliminary exposition of the data supports our use of fixed effects, as we observe distinct temporal and spatial trends in all three mortality measures. The data include 19 years (although 5 election years are omitted in most models such that each county has 14 observations) and 3026 counties. While overall mortality levels differ across men and women, general trends are similar. Figure 1 presents the national average crude rates by COD and sex. For males aged 20–69 years, mortality rates for all COD (right axis) range from approximately 540 to 620 per 100,000, with an increase in recent years. Mortality rates for women of the same age range from 360 to 400 per 100,000. Mortality rates for major cardiovascular COD (left axis) show a slight u shape, with years of decline before a more recent increase.

By comparison, suicide has steadily increased across the years in our sample for both men and women.

The distribution of PL_c is roughly normal, with a mean of 0.48, standard deviation of 0.17, and a range of 0.03–0.97, suggesting that some counties experience drastic PL swings from one administration to another, but most experience a PL_c change of less than 0.34 across elections.

4 Results

Results from Eqs. (1–3) are reported separately for underlying CODs: all-cause, cardiovascular, and suicide. Overall model fit is exceedingly high ($R^2 > 0.9$ for all-cause mortality models), but this is unsurprising given the large set of fixed effects. Results are qualitatively consistent across all CODs and model specifications.

Equations (1–2) estimate the direct relationship between PL_c and mortality. Results are robust across models (Tables 1, 2) and suggest a statistically significant and meaningful relationship. For a 10% increase in PL_c, all-cause mortality will increase by 1% for both men and women (Table 2, column 2).[4] A similar increase is observed across each COD included in the analysis (Table 2, columns 3, 5). While men have significantly higher mortality rates across all CODs, the marginal effects of PL as a percent are similar across sexes. The point estimate for women's mortality caused by cardiovascular events is slightly higher, suggesting that a 10% change in PL_c may lead to a 1.4% increase in cardiovascular-related deaths for females but only a 0.6% increase for males (Table 2, column 4).

---

[4] Marginal effects of dummy variables are calculated using the Halvorsen and Palmquist [93] approximation method described by Bellemare and Wichman [94] and Lim [95].

Table 1 Equation 1

Variables	(1) Total y	(2) Total $\sinh^{-1}(y_{it})$	(3) Cardio. y	(4) Cardio. $\sinh^{-1}(y_{it})$	(5) Suicide y	(6) Suicide $\sinh^{-1}(y_{it})$
Men						
PL	48.54*** (9.68)	0.09*** (0.02)	8.38*** (2.07)	0.07*** (0.01)	1.83*** (0.48)	0.05** (0.02)
Constant	565.03*** (4.67)	6.96*** (0.01)	157.56*** (1.03)	5.64*** (0.01)	26.70*** (0.24)	3.82*** (0.01)
Observations	42,168	42,168	42,168	42,168	42,168	42,168
R^2	0.92	0.89	0.86	0.70	0.44	0.36
Women						
PL	36.42*** (5.52)	0.12*** (0.02)	9.24*** (1.36)	0.15*** (0.02)	0.29 (0.23)	0.04 (0.03)
Constant	346.17*** (2.72)	6.46*** (0.01)	75.26*** (0.68)	4.83*** (0.01)	7.48*** (0.12)	2.37*** (0.02)
Observations	42,168	42,168	42,168	42,168	42,168	42,168
R^2	0.91	0.81	0.83	0.63	0.26	0.38

Robust standard errors in parentheses. Fixed effects are omitted for succinctness

PL partisan loss

***$p < 0.01$, **$p < 0.05$, *$p < 0.1$

Table 2 Equation 2

Variables	(1) Total y	(2) Total $\sinh^{-1}(y_{it})$	(3) Cardio. y	(4) Cardio. $\sinh^{-1}(y_{it})$	(5) Suicide y	(6) Suicide $\sinh^{-1}(y_{it})$
Men						
PL	52.76*** (7.85)	0.10*** (0.01)	9.57*** (1.94)	0.06*** (0.01)	2.74*** (0.51)	0.11*** (0.02)
Constant	562.99*** (3.79)	6.95*** (0.01)	156.99*** (0.95)	5.65*** (0.01)	26.26*** (0.25)	3.79*** (0.01)
Observations	42,168	42,168	42,168	42,168	42,168	42,168
R^2	0.93	0.89	0.86	0.71	0.45	0.37
Women						
PL	32.65*** (4.26)	0.10*** (0.01)	10.16*** (1.18)	0.14*** (0.02)	0.61** (0.24)	0.15*** (0.04)
Constant	347.99*** (2.08)	6.46*** (0.01)	74.81*** (0.58)	4.84*** (0.01)	7.32*** (0.12)	2.32*** (0.02)
Observations	42,168	42,168	42,168	42,168	42,168	42,168
R^2	0.92	0.82	0.84	0.64	0.28	0.40

Robust standard errors in parentheses. Fixed effects are omitted for succinctness

PL partisan loss

***$p < 0.01$, **$p < 0.05$, *$p < 0.1$

Equation (3) allows the effect of PL_d to vary across years within an election cycle. Using winning in the election year as a baseline, a significant effect is observed for PL in each subsequent year. In each year after the election, mortality rates increase with PL. Interestingly, there is no effect on mortality in the election year itself (Table 3).

Inexplicably, we observe a marked decline in suicide for counties that will ultimately lose the election in an election year. While overall results refute earlier work by Classen and Dunn [49], who suggested that election losses reduced suicide, we do observe a similar phenomenon in election years. In election years, counties supporting the eventual loser exhibit a lower mortality rate than counties that support the eventual winner. This result (similar to Classen and Dunn's finding [49]) is perplexing, since 10 months of the election year occur before PL is realized, yet counties that will eventually lose experience a significant decrease in mortality that year. While the unintentional replication of Classen and Dunn's curious finding should be of interest to future researchers, our results suggest that all-cause, cardiovascular, and suicide mortality increase when the entire election cycle is considered.

Table 3 Equation 3

Variables	(1) Total y	(2) Total $\sinh^{-1}(y_{it})$	(3) Cardio. y	(4) Cardio. $\sinh^{-1}(y_{it})$	(5) Suicide y	(6) Suicide $\sinh^{-1}(y_{it})$
Men						
$PL_d \times Y0$	− 3.67 (9.14)	0.00 (0.01)	− 1.58 (2.66)	− 0.01 (0.01)	− 0.65 (0.44)	− 0.01 (0.01)
$PL_d \times Y1$	6.63*** (2.18)	0.01*** (0.00)	0.23 (0.71)	− 0.00 (0.00)	0.41* (0.23)	0.02 (0.01)
$PL_d \times Y2$	12.88*** (2.20)	0.03*** (0.00)	1.95** (0.80)	0.01*** (0.00)	0.48** (0.24)	0.02** (0.01)
$PL_d \times Y3$	6.78*** (2.20)	0.02*** (0.00)	1.44** (0.73)	0.01** (0.00)	0.55** (0.22)	0.02** (0.01)
Constant	586.54*** (1.71)	6.99*** (0.00)	161.52*** (0.52)	5.67*** (0.00)	27.47*** (0.10)	3.83*** (0.00)
Observations	57,228	57,228	57,228	57,228	57,228	57,228
R^2	0.89	0.88	0.83	0.70	0.43	0.36
Women						
$PL_d \times Y0$	− 2.31 (5.91)	0.00 (0.01)	− 0.33 (1.51)	− 0.01 (0.01)	− 0.44*** (0.16)	− 0.03* (0.02)
$PL_d \times Y1$	4.20*** (1.41)	0.01*** (0.00)	1.52*** (0.45)	0.01 (0.01)	− 0.12 (0.12)	0.00 (0.02)
$PL_d \times Y2$	8.07*** (1.45)	0.03*** (0.00)	2.71*** (0.51)	0.04*** (0.01)	0.01 (0.13)	0.03 (0.02)
$PL_d \times Y3$	6.88*** (1.48)	0.02*** (0.00)	2.20*** (0.47)	0.04*** (0.01)	0.13 (0.11)	0.03* (0.02)
Constant	362.25*** (1.10)	6.51*** (0.00)	79.26*** (0.30)	4.89*** (0.00)	7.67*** (0.04)	2.38*** (0.01)
Observations	57,228	57,228	57,228	57,228	57,228	57,228
R^2	0.87	0.81	0.81	0.63	0.27	0.39

Robust standard errors in parentheses. Fixed effects are omitted for succinctness

PL_d discrete partisan loss

***$p < 0.01$, **$p < 0.05$, *$p < 0.1$

4.1 Additional Robustness Tests

Although Eqs. (1–3) all assume a different structural relationship of PL and mortality, consistent qualitative results strongly suggest a significant and meaningful effect. To provide additional robustness checks and to help identify the appropriate structural relationship, three additional approaches are considered.

First, a placebo treatment is created and used in Eq. (2). As a placebo, each election–county cycle is randomly assigned partisan loss scores (PL_c) across the sample and estimated with these placebo scores in place of actual PL scores. While placebo treatments are imprecise, they provide at least some measure that informs the validity of the underlying estimation strategy [96]. For Eq. (2), coefficient estimates on PL placebo treatments prove highly insignificant ($p > 0.1$). Although placebo tests are not conclusive, they bolster the argument that statistically significant PL effects are real and not simply an artifact of a larger dataset or unobserved phenomenon.

Next, PL_c is removed from Eq. (2) such that mortality is regressed only on the state–year and county fixed effects, but no metric of PL. The residuals from this estimation are then plotted against binned PL_c. A strong positive relationship is visible in Fig. 2, which suggests that the exclusion of PL_c from the model leads to an over prediction in counties with low levels of PL and an under prediction in counties with high levels of PL.

Moreover, the continuity of the line—or lack of discontinuity—at 0.5 supports the use of a continuous PL score rather than a discrete loss metric, since at this point a county is evenly divided between winners and losers. Lastly, this figure provides weak evidence that the relationship between PL_c and mortality is not linear, since counties with very low (or high) PL_c are substantially over (or under) estimated when PL_c is excluded from the model. Figure 2 presents a best-fit line, regressing residuals on to PL_c, where
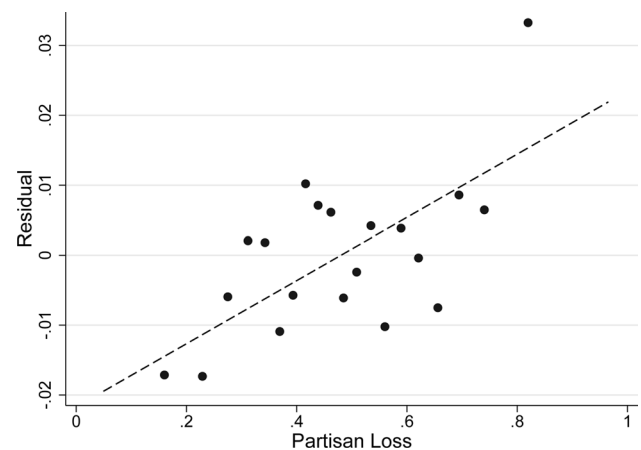

Fig. 2 Residual plot of auxiliary regression. Binned residuals of Ordinary Least Squares model, excluding partisan loss
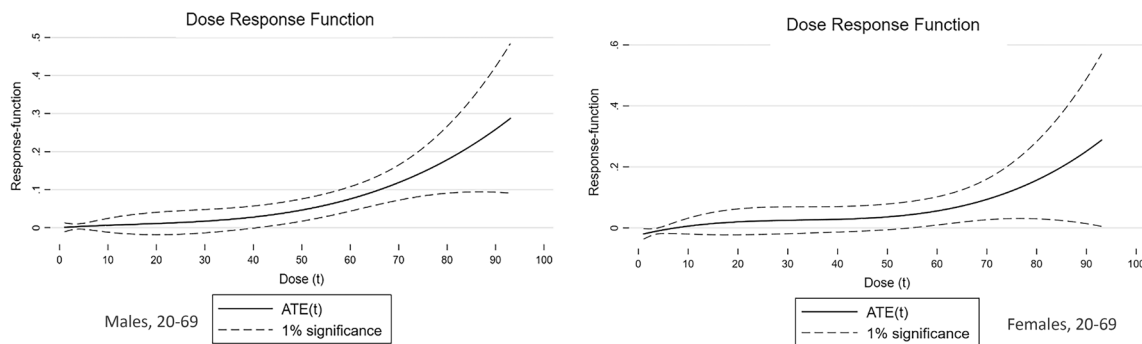
Fig. 3 Partisan loss as a dose–response model. Dose–response average treatment effect (ATE) of partisan loss (scaled from 0 to 100)

the third-order polynomial function provides a slight fit improvement to a simple linear function.

Lastly, PL is treated as a dose–response function, where county–years are "treated" when $PL_d = 1$ and untreated otherwise. The "dose" is the degree of loss (PL_c) normalized between 0 and 100. The full model is omitted for succinctness but identical to the unconfounded model presented by Cerulli [97], where a vector of dummies for county and year are included as covariates. The dose–response is specified as a third-degree polynomial function to allow for changes in the first- and second-order derivatives.

Figure 3 presents predicted mortality responses due to PL, when such losses are assumed to have a "treatment–dose effect." Consistent with models 1–3, mortality increases with PL at an increasing rate. These results suggest the linear assumption of models 1–2 may be erroneous, such that increased mortality may be concentrated in extremely partisan counties (model S2 is included in the ESM and controls for "extreme partisanship" via dummy variables).

5 Discussion

A clear finding from this analysis is the existence of a relationship between partisan election results and mortality rates. The data broadly suggest that mortality rates increase by 0.7% for every 10% of the population that votes for the losing candidate. Considering the large partisan swings in some counties across elections, this effect is meaningful and suggests increased mortality rates of over 3% for extremely partisan counties (where PL may jump from 0.1 to 0.9 from one election to another). Careful interpretation of our results is necessary, as well as a consideration of causality. In this context, there is significant reason to believe that election results are largely exogenous to the individual, such that the response in mortality rates may be properly attributed to PL. However, it is also true that simply losing a partisan election is unlikely to have a direct mechanistic link, such that claiming one causes the other may be misleading. Instead, we posit that losing an

election may influence mortality through some combination of realized partisan policy and social disintegration.

The lack of significance in election years is hypothesized to exist for two reasons, although neither are formally tested herein. The null effect of PL in election years may be a function of the temporal incongruity of our data, where election years have months both before and after an election, and therefore 10 months of an election year where counties support the current election winner may be assigned a losing score. Equally plausible is that election years are fundamentally different since salient hope (or despair) over the impending election will likely interact with any effects of PL. Indeed, there is a long tradition of literature examining the effect of elections and other ceremonial events on mortality, with mixed results [86, 88, 98].

Despite the strength of our statistical results, this study has several limitations. First, we are limited by the data collected and are unable to gain additional insights into individual characteristics and policies that may help elucidate particular mechanisms for mortality rate increases. Second, we are limited to annual data and therefore miss any acute responses to the election, particularly leading up to and following the election. Observing changes immediately following the election may help differentiate the psychosocial effects from the effects of partisan policy, since the election occurs 2 months before a president can affect policy. Lastly, it is possible that the relationship of mortality and voting is bi-directional and non-linear. While the models used in this analysis control for several spatial–temporal fixed effects and rely on relatively short election cycles to determine PL, recent work has suggested that communities that have experienced stagnation or declines in life expectancy in recent decades were significantly more likely to favor Donald Trump in the 2016 election [99, 100].

Combining these findings with previous work, there is legitimate concern that such swings in mortality may also have non-linear feedbacks. For example, Rodriguez et al. [101] noted that excess mortality among the African-American population reduced the 2004 Black voting-age population by approximately 1.7 million, and as much as

3.9 million if incarceration is considered [102]. A similar phenomenon exists among those in poor health [103]. Neiman et al. [104] suggested that traditional "at the polls" voting is more stressful, as measured by increases in cortisol levels, such that mail-in ballots may increase political participation among individuals who are sensitive to social stressors and thus underrepresented in the current voting system. Thus, the models presented herein may be insufficient to capture feedbacks and nuances associated with the mortality–election cycle.

To the extent that the observed mortality response is due to policy changes, our results add to the evidence that median-voter theory does not sufficiently describe political behavior. If few policy differences exist across winning and losing candidates, as median-voter theory suggests, mortality rates should not change based on election outcomes. Although we do not explicitly evaluate the creation of partisan policy, the increase in mortality rates for counties that do not support the president may suggest that such partisanship exists.

To the extent that social cohesion drives the mortality responses identified herein, a presidential candidate may help ameliorate such feelings through messages of unity and bipartisanship. Indeed, discrimination has significant impacts on a range of health outcomes, including blood pressure, cholesterol, body mass index, and self-assessed general health [105]. When combined with previous work, our findings strongly suggest that messages of unity from presidential winners may significantly improve health outcomes by encouraging social cohesion.

While the results presented herein are robust across numerous specifications, there is a significant need to elucidate the relationship of PL, the social determinants of health, and actual health outcomes. Future work should empirically investigate the interaction of national politics and local communities through the lens of social cohesion and rational partisan theory, a topic that is particularly relevant given the increased geographic segregation of liberals and conservatives [36]. This additional work would provide robustness to our own findings and may help with causal interpretation. It may also suggest that the winner-take-all system we use in partisan elections may have significant unacknowledged costs for supporters of the losing candidate, which may be exacerbated by the increased geographic segregation [106–108].

Declarations

Ethics approval Not applicable.

Consent to participate Not applicable.

Consent for publication Not applicable.

Conflict of interest Alexander Maas and Liang Lu have no conflicts of interest that are directly relevant to the content of this article.

Author contributions Both authors contributed to the creation of the relevant model specifications presented herein. Dr. Maas wrote the majority of the paper, and Dr. Lu helped with framing and additional references.

Availability of data and material CDC compressed mortality data can be obtained directly from the CDC with appropriate approval. Dave Leip's Atlas of Presidential Elections is proprietary and can be purchased here: https://uselectionatlas.org/.

Code availability All code used in the analysis will be provided upon request.

References

1. Pew Research Center. Political polarization in the American public. 2014.
2. Iyengar S, Lelkes Y, Levendusky M, Malhotra N, Westwood SJ. The origins and consequences of affective polarization in the United States. Annu Rev Polit Sci. 2019;22:129–46.
3. Green DP, Palmquist B, Schickler E. Partisan hearts and minds: political parties and the social identities of voters. London: Yale University Press; 2004.
4. Ezrow L, Tavits M, Homola J. Voter polarization, strength of partisanship, and support for extremist parties. Comp Polit Stud. 2014;47(11):1558–83.
5. Wallner JI. The death of deliberation: partisanship and polarization in the United States Senate. Washington, DC: Lexington Books; 2013.
6. Muirhead R, Rosenblum NL. Speaking truth to conspiracy: partisanship and trust. Crit Rev. 2016;28(1):63–88.
7. Lau RR, Andersen DJ, Redlawsk DP. An exploration of correct voting in recent US presidential elections. Am J Pol Sci. 2008;52(2):395–411.
8. Polsby NW, Wildavsky AB, Hopkins DA. Presidential elections: strategies and structures of American politics. Washington, Dc: Rowman & Littlefield; 2008.
9. Rodriguez JM, Bound J, Geronimus AT. US infant mortality and the President's party. Int J Epidemiol. 2014;43(3):818–26.
10. Page A, Morrell S, Taylor R. Suicide and political regime in New South Wales and Australia during the 20th century. J Epidemiol Community Health. 2002;10:766–72.
11. Shaw M, Dorling D, Smith GD. Mortality and political climate: how suicide rates have risen during periods of Conservative government, 1901–2000. J Epidemiol Community Health. 2002;723–725.
12. Lee BX, Wexler BE, Gilligan J. Political correlates of violent death rates in the US, 1900–2010: longitudinal and cross-sectional analyses. Aggress Violent Behav. 2014;19(6):721–8.
13. Gentzkow M, Shapiro JM. Ideological segregation online and offline. Q J Econ. 2011;126(4):1799–839.
14. Doherty C. Political polarization in the American public: how increasing ideological uniformity and partisan antipathy affect

politics, compromise and everyday life. Pew Research Center; 2014.

15. Pierce L, Rogers T, Snyder JA. Losing hurts: the happiness impact of partisan electoral loss. J Exp Polit Sci. 2016;3(1):44–59.

16. Marmot M, Allen J, Bell R, Bloomer E, Goldblatt P. WHO European review of social determinants of health and the health divide. Lancet. 2012;380(9846):1011–29.

17. Lucyk K, McLaren L. Taking stock of the social determinants of health: a scoping review. PLoS ONE. 2017;12(5):e0177306.

18. Kahn A, Hottes J, Davis WL. Cooperation and optimal responding in the Prisoner's Dilemma game: effects of sex and physical attractiveness. J Pers Soc Psychol. 1971;17(3):267.

19. Steptoe A, Kivimäki M. Stress and cardiovascular disease. Nat Rev Cardiol. 2012;9(6):360.

20. Kawachi I, Berkman L. Social cohesion, social capital, and health. Soc Epidemiol. 2000;174(7).

21. Verstyuk S. Partisan differences in economic outcomes and corresponding voting behavior: evidence from the US. Public Choice. 2004;120(1–2):169–89.

22. Rose S. Do fiscal rules dampen the political business cycle? Public Choice. 2006;128(3–4):407–31.

23. Reingewertz Y, Baskaran T. Distributive spending and presidential partisan politics. Public Choice. 2019;1–21.

24. Wigley S, Akkoyunlu-Wigley A. Do electoral institutions have an impact on population health? Public Choice. 2011;148(3–4):595–610.

25. Bolton A, Potter RA, Thrower S. Organizational capacity, regulatory review, and the limits of political control. J Law Econ Organ. 2016;32(2):242–71.

26. Howell WG. Introduction: unilateral powers: a brief overview. Pres Stud Q. 2005;35(3):417–39.

27. Wilkinson RG. Income inequality, social cohesion, and health: clarifying the theory—a reply to Muntaner and Lynch. Int J Health Serv. 1999;29(3):525–43.

28. Wilkinson RG, Kawachi I, Kennedy BP. Mortality, the social environment, crime and violence. Sociol Health Illn. 1998;20(5):578–97.

29. Mulvaney-Day NE, Alegria M, Sribney W. Social cohesion, social support, and health among Latinos in the United States. Soc Sci Med. 2007;64(2):477–95.

30. Hawe P, Shiell A. Social capital and health promotion: a review. Soc Sci Med. 2000;51(6):871–85.

31. Muennig PA, Reynolds M, Fink DS, Zafari Z, Geronimus AT. America's declining well-being, health, and life expectancy: not just a white problem. Am J Public Health. 2018;108(12):1626–31.

32. Elderon L, Whooley MA. Depression and cardiovascular disease. Prog Cardiovasc Dis. 2013;55(6):511–23.

33. Rodwin BA, Spruill TM, Ladapo JA. Economics of psychosocial factors in patients with cardiovascular disease. Prog Cardiovasc Dis. 2013;55(6):563–73.

34. Reidpath DD. "Love thy neighbour"—it's good for your health: a study of racial homogeneity, mortality and social cohesion in the United States. Soc Sci Med. 2003;57(2):253–61.

35. Inoue S, Yorifuji T, Takao S, Doi H, Kawachi I. Social cohesion and mortality: a survival analysis of older adults in Japan. Am J Public Health. 2013;103(12):e60–6.

36. Motyl M, Iyer R, Oishi S, Trawalter S, Nosek BA. How ideological migration geographically segregates groups. J Exp Soc Psychol. 2014;51:1–14.

37. Ladner M, Wlezien C. Partisan preferences, electoral prospects, and economic expectations. Comp Polit Stud. 2007;40(5):571–96.

38. Puri M, Robinson DT. Optimism and economic choice. J Financ Econ. 2007;86(1):71–99.

39. Keele L. The authorities really do matter: party control and trust in government. J Polit. 2005;67(3):873–86.

40. Sheffer L. Partisan in-group bias before and after elections. Elect Stud. 2020;67:102191.

41. Waismel-Manor I, Ifergane G, Cohen H. When endocrinology and democracy collide: emotions, cortisol and voting at national elections. Eur Neuropsychopharmacol. 2011;21(11):789–95.

42. Stanton SJ, Beehner JC, Saini EK, Kuhn CM, LaBar KS. Dominance, politics, and physiology: voters' testosterone changes on the night of the 2008 United States presidential election. PLoS ONE. 2009;4(10):e7543.

43. Blanton H, Strauts E, Perez M. Partisan identification as a predictor of cortisol response to election news. Polit Commun. 2012;29(4):447–60.

44. Williams DR, Mohammed SA. Racism and Health II: a needed research agenda for effective interventions. Am Behav Sci. 2013;57(8).

45. Morey BN. Mechanisms by which anti-immigrant stigma exacerbates racial/ethnic health disparities. Am J Public Health. 2018;108(4):460–3.

46. Hatzenbuehler ML, Prins SJ, Flake M, Philbin M, Frazer MS, Hagen D, et al. Immigration policies and mental health morbidity among Latinos: a state-level analysis. Soc Sci Med. 2017;174:169–78.

47. Gemmill A, Catalano R, Casey JA, Karasek D, Alcalá HE, Elser H, et al. Association of preterm births among US Latina Women With the 2016 Presidential Election Preterm Births Among US Latina Women After the 2016 Presidential Election. JAMA Netw Open. 2019;2(7):e197084–e197084.

48. Orr ST, James SA, Blackmore PC. Maternal prenatal depressive symptoms and spontaneous preterm births among African–American Women in Baltimore, Maryland. Am J Epidemiol. 2002;156(9):797–802.

49. Classen TJ, Dunn RA. The politics of hope and despair: the effect of presidential election outcomes on suicide rates. Soc Sci Q. 2010;91(3):593–612.

50. Chopik WJ, Motyl M. Ideological fit enhances interpersonal orientations. Soc Psychol Pers Sci. 2016;7(8):759–68.

51. Citrin J, Stoker L. Political trust in a cynical age. Annu Rev Polit Sci. 2018;21:49–70.

52. Yonk RM. Politics and quality of life the role of well-being in political outcomes. New York: Springer; 2018.

53. Gonzalez KA, Ramirez JL, Galupo MP. Increase in GLBTQ minority stress following the 2016 US presidential election. J GLBT Fam Stud. 2018;14(1–2):130–51.

54. Kauder B, Potrafke N, Reischmann M. Do politicians reward core supporters? Evidence from a discretionary grant program. Eur J Polit Econ. 2016;45:39–56.

55. Potrafke N, Roesel F. The urban–rural gap in healthcare infrastructure: does government ideology matter? Reg Stud. 2019;1–12.

56. Tufte ER. Political control of the economy. Princeton University Press; 1978.

57. Coibion O, Gorodnichenko Y. Is the Phillips curve alive and well after all? Inflation expectations and the missing disinflation. Am Econ J Macroecon. 2015;7(1):197–232.

58. Carlsen F. Rational partisan theory: empirical evidence for the United States. South Econ J. 1998;64–82.

59. Cox GW, McCubbins MD. Electoral politics as a redistributive game. J Polit. 1986;48(2):370–89.

60. Curto-Grau M, Solé-Ollé A, Sorribas-Navarro P. Does electoral competition curb party favoritism? Am Econ J Appl Econ. 2018;10(4):378–407.

61. Harris JA, Posner DN. (Under What Conditions) Do politicians reward their supporters? Evidence from Kenya's Constituencies Development Fund. Am Polit Sci Rev. 2019;113(1):123–39.

62. Franzese RJ Jr. Electoral and partisan cycles in economic policies and outcomes. Annu Rev Polit Sci. 2002;5(1):369–421.

63. Hibbs D. The American political economy: electoral policy and macroeconomics in contemporary America. Cambridge: Harvard University; 1987.

64. Downs A. An economic theory of democracy. New York: Harper; 1957.

65. Alesina A. Macroeconomics and politics. NBER Macroecon Ann. 1988;3:13–52. https://doi.org/10.1086/654070

66. Hibbs DA Jr. Partisan theory after fifteen years. Eur J Political Econ. 1992;8(3):361–73.

67. Gerber ER, Lewis JB. Beyond the median: Voter preferences, district heterogeneity, and political representation. J Political Econ. 2004;112(6):1364–83.

68. Courtemanche C, Marton J, Ukert B, Yelowitz A, Zapata D. Early impacts of the affordable care act on health insurance coverage in Medicaid expansion and non-expansion states. J Policy Anal Manag. 2017;36(1):178–210.

69. Barbaresco S, Courtemanche CJ, Qi Y. Impacts of the Affordable Care Act dependent coverage provision on health-related outcomes of young adults. J Health Econ. 2015;40:54–68.

70. Bailey MJ, Goodman-Bacon A. The War on Poverty's experiment in public medicine: Community health centers and the mortality of older Americans. Am Econ Rev. 2015;105(3):1067–104.

71. Card D, Dobkin C, Maestas N. The impact of nearly universal insurance coverage on health care utilization: evidence from Medicare. Am Econ Rev. 2008;98(5):2242–58.

72. Montez JK, Beckfield J, Cooney JK, Grumbach JM, Hayward MD, Koytak HZ, et al. US State Policies, politics, and life expectancy. Milbank Q. 2020;98(3):668–99.

73. Bowleg L. We're not all in this together: On COVID-19, intersectionality, and structural inequality. Am J Public Health. 2020;110(7):917.

74. Stephenson J. COVID-19 outbreaks among food production workers may intensify pandemic's disproportionate effects on people of color. JAMA Health Forum. 2020;e200783–e200783.

75. Ellig J, Conover CJ. Presidential priorities, congressional control, and the quality of regulatory analysis: an application to healthcare and homeland security. Public Choice. 2014;161(3–4):305–20.

76. Gordon SC, Landa D. Polarized preferences versus polarizing policies. Public Choice. 2018;176(1–2):193–210.

77. Kang HK, Bullman TA. Mortality among US veterans of the Persian Gulf War. N Engl J Med. 1996;335(20):1498–504.

78. Lester D. The effect of war on suicide rates. Eur Arch Psychiatry Clin Neurosci. 1993;242(4):248–9.

79. Osterberg D, Wallinga D. Addressing externalities from swine production to reduce public health and environmental impacts. Am J Public Health. 2004;94(10):1703–8.

80. Aguilera N, Marrufo GM. Can better infrastructure and quality reduce hospital infant mortality rates in Mexico? Health Policy. 2007;80(2):239–52.

81. Sperling JB, Ramaswami A. Exploring health outcomes as a motivator for low-carbon city development: implications for infrastructure interventions in Asian cities. Habitat Int. 2013;37:113–23.

82. Huberman G, Konitzer T, Krupenkin M, Rothschild D, Hill S. Economic expectations, voting, and economic decisions around elections. AEA Papers and Proceedings. 2018. p. 597–602.

83. Dangol J. Unanticipated political events and stock returns: an event study. Econ Rev. 2008;20:86–110.

84. Al-Ississ MM, Miller NH. What does health reform mean for the health care industry? Evidence from the Massachusetts special senate election. Am Econ J Econ Policy. 2013;5(3):1–29.

85. Brainerd E. Economic reform and mortality in the former Soviet Union: a study of the suicide epidemic in the 1990s. Eur Econ Rev. 2001;45(4–6):1007–19.

86. Phillips DP, Feldman KA. A dip in deaths before ceremonial occasions: Some new relationships between social integration and mortality. Am Sociol Rev. 1973;678–96.

87. Schulz R, Bazerman M. Ceremonial occasions and mortality: a second look. Am Psychol. 1980;35(3):253.

88. Boor M. Reduction in deaths by suicide, accidents, and homicide prior to United States presidential elections. J Soc Psychol. 1982;118(1):135–6.

89. Wasserman IM. Political business cycles, presidential elections, and suicide and mortality patterns. Am Sociol Rev. 1983;711–720.

90. Wolfers J. Did unilateral divorce laws raise divorce rates? A reconciliation and new results. Am Econ Rev [Internet]. 2006;96(5):1802–20.

91. Afzal M. Do barriers to candidacy reduce political competition? Evidence from a bachelor's degree requirement for legislators in Pakistan. Public Choice. 2014;161(1–2):51–72.

92. Solon G, Haider SJ, Wooldridge JM. What are we weighting for? J Hum Resour. 2015;50(2):301–16.

93. Halvorsen R, Palmquist R. The interpretation of dummy variables in semilogarithmic equations. Am Econ Rev. 1980;70(3):474–5.

94. Bellemare MF, Wichman CJ. Elasticities and the inverse hyperbolic sine transformation. Oxf Bull Econ Stat. 2020;82:50–61.

95. Lim JJ. The limits of central bank independence for inflation performance. Public Choice. 2020;2:1–27.

96. Clemens J. Pitfalls in the Development of Falsification Tests: an illustration from the Recent Minimum Wage Literature. 2017.

97. Cerulli G. ctreatreg: Command for fitting dose–response models under exogenous and endogenous treatment. Stata J. 2015;15(4):1019–45.

98. Shimizu M, Pelham BW. Postponing a date with the grim reaper: ceremonial events and mortality. Basic Appl Soc Psychol. 2008;30(1):36–45.

99. Bor J. Diverging life expectancies and voting patterns in the 2016 US presidential election. Am J Public Health. 2017;107(10):1560–2.

100. Bilal U, Knapp EA, Cooper RS. Swing voting in the 2016 presidential election in counties where midlife mortality has been rising in white non-Hispanic Americans. Soc Sci Med. 2018;197:33–8.

101. Rodriguez JM, Geronimus AT, Bound J, Dorling D. Black lives matter: differential mortality and the racial composition of the US electorate, 1970–2004. Soc Sci Med. 2015;136:193–9.

102. Cottrell D, Herron MC, Rodriguez JM, Smith DA. Mortality, incarceration, and African American disenfranchisement in the contemporary United States. Am Polit Res. 2019;47(2):195–237.

103. Pacheco J, Ojeda C. A healthy democracy? Evidence of unequal representation across health status. Polit Behav. 2019;1–23.

104. Neiman J, Giuseffi K, Smith K, French J, Waismel-Manor I, Hibbing J. Voting at home is associated with lower cortisol than voting at the polls. PLoS ONE. 2015;10(9):e0135289.

105. Johnston DW, Lordan G. Discrimination makes me sick! An examination of the discrimination–health relationship. J Health Econ. 2012;31(1):99–111.

106. Dasgupta I, Neogi RG. Between-group contests over group-specific public goods with within-group fragmentation. Public Choice. 2018;174(3–4):315–34.

107. Martin GJ, Webster SW. Does residential sorting explain geographic polarization? Polit Sci Res Methods. 2018;1–17.

108. Kaplan E, Spenkuch JL, Sullivan R. Measuring geographic polarization: theory and long-run evidence. 2019.