Does Democracy Matter for Lifespan Inequalities? Regime Type and Premature Mortality by Sex

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Abstract: Reductions in premature mortality are widely attributed to economic, educational, and medical factors. This study contributes to our understanding of the influence of political factors in preventing early death and gender inequalities in health outcomes. We analyze data from life tables of the World Health Organization, 2000-2015, to estimate the annual, sex-specific standard deviation of the age-at-death distribution across 162 countries. We apply dynamic panel model analyses to assess the association between political liberalization and inequalities in premature mortality. Our findings show reduced inequalities in premature mortality in liberal democracies, with men benefiting disproportionately. We theorize that liberal democracy may motivate governments to respond to citizens' desires for policies that improve health and reduce risks. As democratic liberalization increases, premature mortality falls for men, which may be accounted for in part by reduced male mortality from injuries. Reductions in premature mortality for women appear to stem primarily from improvements in maternal mortality across regime types. Our findings support the idea that democratization may provide public health benefits, especially for male citizens.

Keywords: gender inequality, democracy, premature mortality, global health.

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Introduction

Global health outcomes have significantly improved in recent decades, resulting in reduced premature mortality rates. Global child mortality, for example, has dropped by 60 percent since 1990 (World Health Organization 2022). Adult life expectancy has also improved globally for both men and women. Maternal mortality associated with pregnancy-related risks fell markedly, resulting in increased overall female life expectancy (World Bank 2023). Yet around the world there remain enormous gaps both within and across countries in health outcomes such as premature mortality (Rajaratnam et al. 2010; Kuhn 2010; Rodriguez et al. 2015).

Improvements in premature mortality—deaths occurring before an individual's expected number of life-years—are widely attributed to economic, educational, technological, and medical factors (McKeown and Record 1962; Caldwell 1986; Wilmoth 2007). Our analysis contributes to a relatively small yet growing literature bringing insights from comparative politics into research in public health. We argue that democratic liberalization reduces inequalities in premature mortality of adults, particularly male adults. Key democratic processes such as political representation and accountability may enable individuals to elect leadership and engage in collective action to affect policy action that may put them at lower risk of premature death in adulthood. In particular, we suggest that sex-specific causes of premature death, such as fatal injuries and accidents, may improve with democratic liberalization.

Recent scholarship has questioned the long-standing view that democracies are more likely to enable healthy and longer lives (Shandra et al. 2004, Burroway 2016). A wide literature shows beneficial effects of democracy on health, but several studies show no effect (Ross 2006; Mejia 2022). Much of what we know about the political factors influencing mortality comes from studies of infant and child mortality, life expectancy at birth, or maternal welfare

(Rodriguez et al. 2022). For example, out of the 201 studies on democracy and population health reviewed by McGuire (2020), only four studies specifically explore adult mortality outcomes. Existing studies predominantly employ measures capturing the premature mortality of infants and young children, or central-tendency measures like the life expectancy at birth. We examine a different indicator, variation in premature mortality in adults, to consider alternative ways that regime type may matter for health outcomes over the course of life.

We use the standard deviation of the age-at-death distribution (S_{20}) to measure variation in premature mortality of the adult population, for both men and women. An increasing standard deviation of age at death signals more deaths at younger adult ages, indicating increased inequality in lifespan. A decreasing standard deviation signals more deaths at older ages and less lifespan inequality, with a higher fraction of the population dying at older ages. Premature death is not evenly distributed across populations within and across nations (Rodriguez et al. 2014). Those with more economic, social, and political resources are better able to avoid risky circumstances such as dangerous occupations, poor infrastructure, exposure to pollutants, stress, unhealthy diets, and unhealthy behaviors that increase their risk of death (Geronimus et al. 2019). As these individuals show high rates of survival, they are also able to influence democratic processes for longer periods of their life (Rodriguez 2018). By exploring premature mortality in young adults and the middle aged, we can infer policy responsiveness to the most economically productive and politically active portion of nations' populations. We ask whether premature mortality differs across levels of liberal democracy and, if so, whether these differences manifest differently for men and women.

We find that as nations increase their level of liberal democracy, they manifest a lower standard deviation in the age at death, even after accounting for levels of economic development, overall levels of mortality, and a battery of controls for endogenous processes. Liberal democracies appear to reduce lifespan inequalities primarily by lowering premature mortality in men (Mackenbach et al. 2013; Espelt et al. 2008; Wejnert 2008). Women do not show large, statistically significant differences in their age-at-death variation across regime types, even if the effect of democracy on variation in mortality is negative for women (i.e., it decreases the standard deviation of age at death), on average. We consider whether these differences by sex point toward possible mechanisms whereby regime type impacts inequalities in premature mortality. Democracies may be particularly likely to improve regulatory policies to reduce injuries, from occupational safety, road injury, and accidents, which disproportionately affect men (Bollyky et al. 2019; Scholtz 1991; Kruk et al. 2018, Winslow 2005). In contrast, women's health inequalities are generally reduced more as a function of the expansion of health care services and preventive care. Liberal democracy may enable collective action or electoral processes to reduce premature mortality via policies that result in lower incidence of injury, beyond larger gains in healthcare, which benefit both men and women but particularly men.

Our research contributes to the understanding of potential mechanisms whereby democratic processes may improve living standards, beyond levels of economic development. We also consider if electoral mechanisms and the legal exercise of collective action may provide channels to improve health and safety for the broad population. Our emphasis on risk and safety regulations also distinguishes our work from public health research focused primarily on healthcare systems. Possible implications of our study include that health gains and losses driven

by democratization may come particularly in the form of differential exposure to physical and other risks.

By combining insights from comparative politics, public health, and social epidemiology, we contribute to research in comparative global health. The use of the standard deviation of age at death is novel to comparative politics. In international epidemiological studies it has been shown to capture health inequalities in a broad, consistent, and coherent cross-national framework (van Raalte et al. 2011; Edwards and Tuljapurkar 2005). Our focus on differential effects of regime type on health inequalities by sex is also novel to comparative politics, to our knowledge. Our study also fits within the public health tradition, asking whether features of democracy affect health equities with an interdisciplinary approach focused on potential causal mechanisms beyond medical determinants of health that may inform policies to increase health equity (Porta 2014).

We are also careful to address the methodological concerns of studying health inequalities in countries of very different levels of economic development and governmental structure. Estimating the impact of regime type on health outcomes is fundamentally challenged with concerns of endogeneity driven by the underlying relationship between economic development and democracy on the one hand, and economic development and health outcomes on the other. Health outcomes improve as countries get richer (Pritchett and Summers 1996; Weil 2014) and countries get richer as their populations get healthier (Bloom et al. 2004; Well 2007). Moreover, countries are more likely to be democracies when they are richer (Przeworski et al. 2000), and democratic factors have been shown to have indirect effects on economic output (Doucouliagos and Ulubaşoğlu 2008). Similarly, health outcomes including premature mortality have been shown to be a cause and effect of political processes (Rodriguez et al. 2015). We

address these issues of endogeneity in our conceptual approach, examining outcome differences between the sexes to identify possible mechanisms of regime effects. We also place endogeneity at the forefront of our empirical approach with system generalized method of moments (SGMM) estimators.

Liberal Democracy and Health Outcomes

The literature in political science and epidemiology holds that democratic regimes are positively associated with improved health outcomes (overall levels and inequalities). Democratic regimes are found to be more active in producing health policy, delivering public health interventions, and institutionalizing health care systems. Among the espoused benefits of democracy are accelerated improvements in non-communicable diseases and injuries (Bollyky et al. 2019). Democracies also appear to reduce health inequalities across key factors such as gender and socioeconomic indicators (Bambra 2013).

Research arguing democracy improves public health emphasizes the role of electoral mechanisms, which may incentivize politicians to provide broader healthcare goods and services than in autocracies (Lake and Baum 2001; Huber and Stephens 2012; Wang, Mechkova and Andersson 2019; McGuire 2010). For example, Wigley and Akkoyunlu-Wigley (2017) argue that democratic leaders have greater motivation than autocratic leaders to reduce child mortality due to the electoral and economic benefits associated with improved infant health. Reeves and Mackenbach (2019) show that political participation is also strongly associated with lower health inequalities. Grépin and Dionne (2013) demonstrate that universal healthcare coverage is a visible policy issue generating high electoral dividends. Shandra et al. (2010) argue that democracy is a pre-condition for the effectiveness of international health and women's organizations' efforts to reduce infant mortality. Pieters et al. (2016) found that a democratic

transition results in a decline in under-five mortality, with the effect increasing over time. If we think democracies are responsive to large groups, health is an evident policy area in which governments could improve life standards and its provision should substantially reduce health inequalities across the population (Mackenbach et al.2013).

Liberal democracies generally outperform autocracies in healthcare outcomes, with the benefits tending to accrue in high-income countries (Besley and Kudamatsu 2006). High-income liberal democracies have higher levels of redistribution, specifically in areas of healthcare and key social determinants of health such as education. A key argument is that, when people vote, they tend to vote for policies that help them as individuals, via human capital, rather than other types of capital that may also boost economic growth (Lake and Baum 2001). Moreover, elections provide incentives to extend human capital to the less fortunate to capture votes (Baum and Lake 2003; Stasavage 2005; Gerring, Thacker, and Alfaro 2012). Kavanaugh (2016) argues that deliberation within liberal democracies also makes it more likely that citizens view health care as a human right and responsibility of the government. As a result, liberal democracies may enjoy more effective healthcare systems and lower health inequalities than non-democratic regimes at similar levels of economic development (Kuhn 2010; Ghobarah, Huth, and Russett 2004; Muntaner et al. 2011).

A related mechanism whereby democracy may matter for population health is through greater collective action opportunities (Bermeo and Yashar 2016). Political parties and civil society organizations are important stakeholders for improving human development. They serve as an informational pathway, imparting the needs of local communities, and putting pressure on politicians to improve health policies. Bernhard et al. (2019) emphasize the importance of

collective action, arguing that political parties and social movements play a vital deterrent role to check political actors who might consider deviating from democratic agreements.

Collective action and direct participatory processes can lead to improved social welfare and health outcomes. Strong rule of law and constitutionally guaranteed civil rights and liberties are important for understanding the health effects of democratic engagement and functioning of democracy (Wigley and Akkoyunlu-Wigley 2011; Cheibub, Hong, and Przeworski 2020). Greer et al. (2017) show how in the early 1990s, civil society in newly democratic Poland pressured the government to adopt significantly improved standards of obstetric care and childbirth procedures. The success of Polish women was facilitated by a free and independent media. In Brazil, community organizations and social advocates pressed the judicial and legislative systems to achieve universal coverage for AIDS testing and treatment (Rich 2019). Research in India shows significant direct health benefits to the poor, especially for women, through local public participation institutions called *gram sabhas* and *gram panchayat* (Gibson 2012; Hamal et al. 2018).

Democratic processes of public engagement may encourage policy feedback that allows voters to influence healthcare policies. Citing examples from Thailand, Chile, and Costa Rica, McGuire (2010) shows reduced mortality (via improved regulations) and improved healthcare services that resulted from constituent pressure on government. The WHO found similar dynamics in the Philippines, Central America, and South Africa (World Health Organization 2007).

The need for an engaged civil society is emphasized during major health crises (Acharya et al. 2020). Epidemics and pandemics bring public health issues to the national level and mobilize citizens to demand an effective government response. Community groups and other

civil society organizations have been important stakeholders in governmental responses to HIV/AIDS in Africa and Latin America (Björkman and Svensson 2009; Wamai 2014). Boone and Batsell (2001) suggest that political liberalization contributed to the effectiveness of policies responding to HIV/AIDS. Senegal and Uganda saw grassroots activism and awareness campaigns build broad legitimacy for AIDS prevention.

Yet research on regime type and health outcomes is not unified. Evidence of the link between democracy and child health is mixed, with some studies finding negative associations between health and democracy (for a review, see Mejia 2022). Some research finds that factors like economic development, improved sanitation, and educational attainment are more important as determinants of child health outcomes than regime type (Shandra et al. 2004, Burroway 2016). Dionne (2011) shows that time horizons, whether in democratic or autocratic regimes, explained government responses to AIDS in Africa.

The effects of democracy on health, if any, may also be indirect. Shandra et al. (2004) find no direct effects from democracy on infant mortality in developing countries, but their results suggest that democracy might have a moderating effect, in which international dependency might negatively affect infant mortality at lower levels of democracy than at higher levels. Similarly, Noble (2019) finds no direct impact of democracy on infant mortality, but instead identifies a mediation effect: democracy increases public health spending, which is positively associated to socio-health resources, which in turn reduces infant mortality.

Health Inequalities by Sex and Regime Type

Our research contributes to the study of regime type on health outcomes as evidenced by gender disparities. Mackenbach et al.(2013) show that in Europe all-cause mortality among men decreases as democracies mature, but the democratization effect does not appear to reduce

mortality among women. Similarly, Wejnert (2008) shows that women in developing countries did not enjoy improved maternal health, labor force participation, and educational opportunities from democratic advancement between 1970 and 2000. Espelt et al. (2008) find that health inequalities in democracies are particularly prevalent for women.

Causes of death vary by sex and levels of economic development. Women are more likely than men to die of communicable diseases, and due to the sex-specific mortality risks associated with pregnancy, birth, and post-partum conditions. The World Bank estimates that about 50% of women around the world who die between ages 15 and 34 do so from communicable diseases (World Bank 2016). Women in this age range are also vulnerable to chronic diseases, such as diabetes, cancer, and cardiovascular illness, as well as domestic violence, with the latter greatly underreported (Krantz 2002; Raymond, Greenberg and Leeder 2005). In contrast, about half of men who die prematurely (between ages 15-34) succumb to injuries from accidents, injuries, interpersonal violence, and self-harm (World Bank 2016). These sex differences suggest how liberal democratic governance may possibly reduce premature mortality among men in particular, by advancing public health policies to regulate hazardous workplaces, improve road infrastructure, and reduce the overall risk of injury.

Democracy may foster social and institutional conditions conducive for collective action to seek public health protections. High-quality healthcare can ensue from political and legal accountability through laws and regulations, transparency, and greater political activism demanding improved working and living conditions. Workplace safety regulation is one of the areas in which we should see governments' role in impacting mortality, particularly for men in the labor force who are disproportionately likely to die of injuries. Cross-national data on occupational mortality shows that its rate in non-democracies is about 30 percent higher than in

democracies (ILO, 2003). Democratic liberalization may also improve health outcomes and health inequalities if safety conditions are improved for a large percentage of the working population, especially as men are most likely to be engaged in the primary and secondary economic sectors in which injuries are particularly likely. Democracies also feel pressure to reduce environmental harm, including those that contribute to cancers and other non-communicable diseases (Winslow 2005).

As democratic liberalization increases, demands tend to rise for safer work and living conditions and a higher quality of life (Inglehart and Welzel 2005; Dorman 1996). For example, Bollyky et al. (2019) show that democracies attenuate the conditions that lead to injury. They estimate that democratic experience is associated with an 18-percent reduction in transportation injuries. Kruk et al. (2018) show that road injury deaths are also more likely to be averted by higher quality care offered in democracies when accidents do occur.

In industrialized nations, women live longer than men (Nathanson 1975; Read and Gorman 2010). Women's health improves with economic development, and as healthcare services and sanitation reduce maternal mortality in particular, women live longer, reducing variation in premature death amongst women. Goldin and Lleras (2019) show a sharp reduction in infectious diseases in the early 20th century contributed to improved female longevity in the United States and Europe. Despite living longer and faring better than men on the leading causes of death, women also experience more illness and comorbidity (communicable and non-communicable), especially at younger ages (Singh-Manoux et al. 2008). Factors impacting female mortality are primarily related to biological, behavioral, and socioeconomic factors (Mackenbach et al. 2008; Read and Gorman 2010; Freeman et al. 2020). Caldwell (1986) credits improvements in female empowerment and education for reduced female mortality and better

health policies in poor nations. These may not be so readily improved in early stages of democratic development or may not differ drastically from those in less democratic societies. Overall, existing research suggests that political mechanisms may improve health outcomes disproportionately for men relative to women.

The Standard Deviation of Age at death

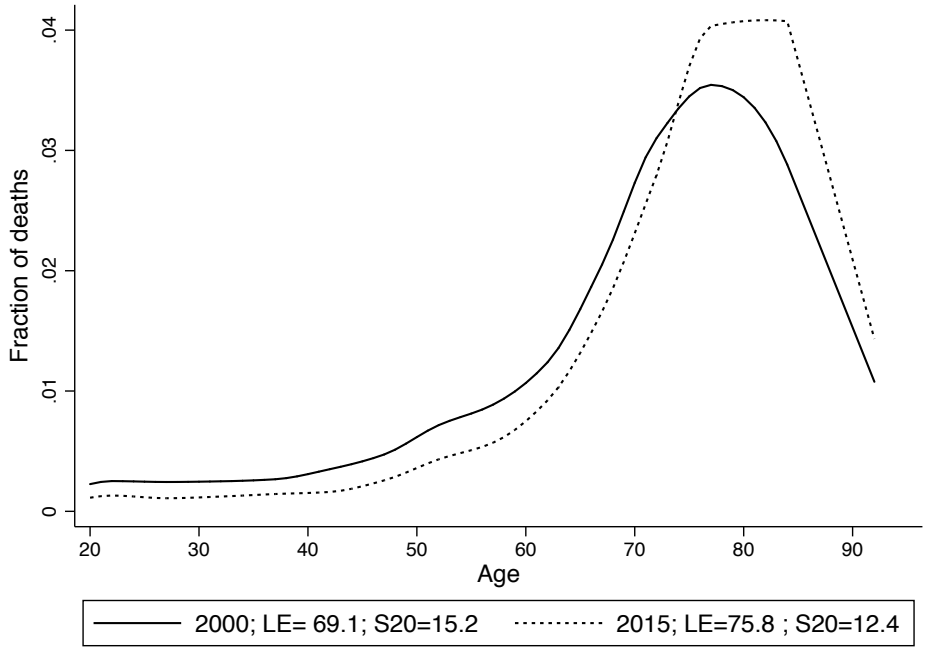
Our analysis employs the standard deviation of the age-at-death distribution conditional on survival to age 20 (S_{20}). This measure captures inequalities in premature death among adults, and is useful for assessing and describing heterogeneity of a population's wellbeing (Sasson 2016). In our study, S_{20} is a measure of the dispersion of proportions of individuals dying at different ages around the central tendency of the human lifespan in a given year and country. Accordingly, S_{20} captures the uncertainty of the length of life for adult populations exposed to country-specific characteristics, including political and economic ones. A larger value of S_{20} implies greater heterogeneity of the underlying factors that determine human longevity.

Our S_{20} indicator is calculated from the full distribution of period age-specific mortality rates above the age of 19. This is the age span in which most political activity, both electoral and nonelectoral, and mortality manifest. Given the left-skewed nature of the age-at-death distribution, with higher fractions of the population dying at older ages, S_{20} is particularly sensitive to deaths occurring before the central tendency of the age at death. Much of the inequality in mortality is due to early, preventable deaths and not due to mortality at older ages. These longevity benefits are parallel to the benefits to life expectancy at birth from reducing infant mortality.

Figure 1 provides an example of the age-at-death distribution for men in Iran. As the age-at-death distribution is compressed from 2000 to 2015, so is its standard deviation. The S_{20}

decreases from 15.2 years to 12.4 years, with life expectancy at birth increasing from 69.1 years in 2000 to 75.8 years in 2015. In Iran, we observe that the decrease in S_{20} during the 15-year period is mostly due to averting premature deaths among adults, with an increased proportion of individuals in 2015 dying after the age where the two distributions intersect.

Figure 1: Age-at-death Distribution, Males, Iran, 2000 and 2015



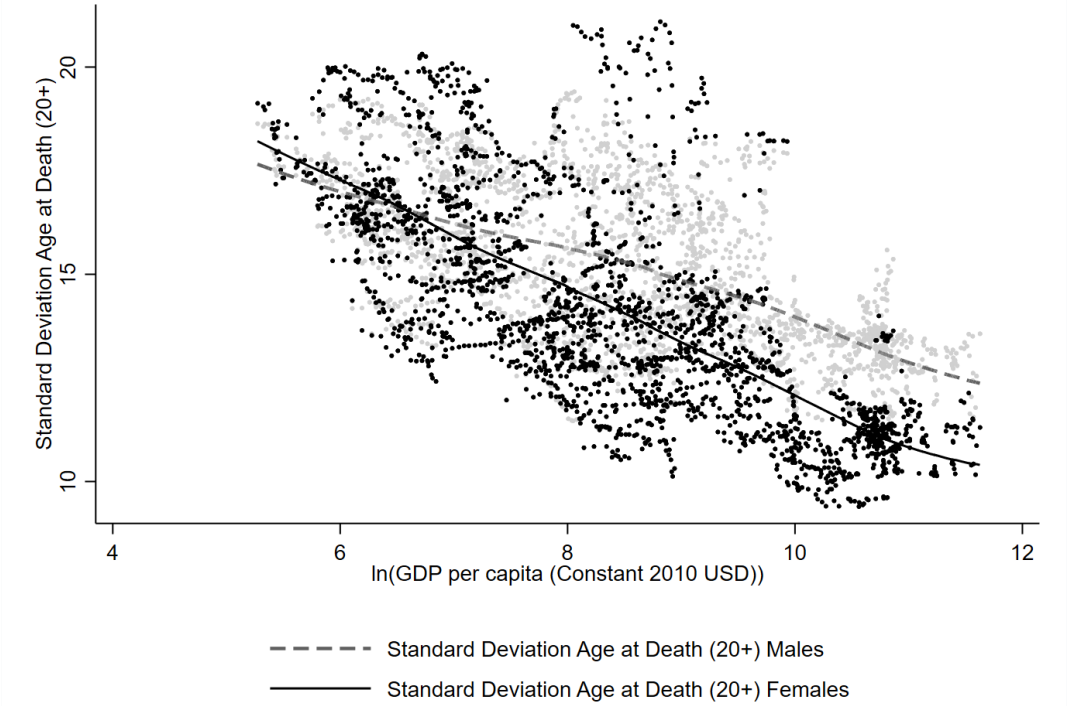
Note: LE refers to life expectancy at birth; S_{20} refers to the standard deviation of age at death conditional on survival to age 20.

As critical factors for survival become similar across adults, reduction in standard deviation of age at death should ensue. Reduction in S_{20} reflects the convergence of mortality scenarios that homogenize in populations, including political, economic, and social sources of variation in longevity. Low levels of variability in the age of death are particularly evident in economically advanced industrial nations, which are mostly democracies. These countries have high life expectancy at birth, relative to developing nations, and are potentially approaching the

biological limit of the human lifespan (Fries 1980; Olshansky, Carnes and Cassel 1990; Wilmoth 2007; Vallin and Meslé 2010). Less developed nations, however, are likely to experience larger gains in life expectancy at birth, considering that a larger proportion of their population is still far away from the life expectancies experienced in advanced industrial democracies. While the age-at-death distribution remains relatively stable in advanced industrialized nations and middle income nations in the last decades, S_{20} is shrinking in the less developed world.

This situation is illustrated in Figure 2. The S_{20} shrinks as countries become richer. This is particularly the case for women, for whom economic development appears to mitigate health risks for premature death, especially maternal conditions (Roser and Ritchie 2013; Wang 2014). The S_{20} falls for men as well as development increases, but to a lesser extent than for women. As the level of economic development increases, the sex difference in S_{20} also increases, with lower premature mortality among women relative to men.

Figure 2. Standard Deviation of Age-at-death and GDP per Capita by Sex



Note: All countries for all years (2000-2015) included.

Another implication of these observations on mortality variation is that typical central tendency indicators of population well-being and longevity, such as life expectancy at birth, may remain relatively stable over long periods of time. In contrast, the S_{20} measure is more sensitive to variation arising from underlying mortality mechanisms within and across nations. Mortality inequality is primarily due to differences in the age at death among adults in wealthier countries (Tuljapurkar 2010). Central tendency measures of longevity may remain static in spite of increasing heterogeneity in death among middle-age and older adults, thus masking variation in underlying processes. Edwards and Tuljapurkar (2005) hold mortality displayed in life-tables should not only be characterized by the mean, or other measures of central tendency, but equally important by measures of the variability in the distribution of mortality rates. S_{20} therefore allows us to map variation—not linked to any particular age—that could not be otherwise captured by purely chronological age indicators of the lifespan (Kannisto 2000, Sasson 2016).

Data

Standard Deviation of Age-at-death

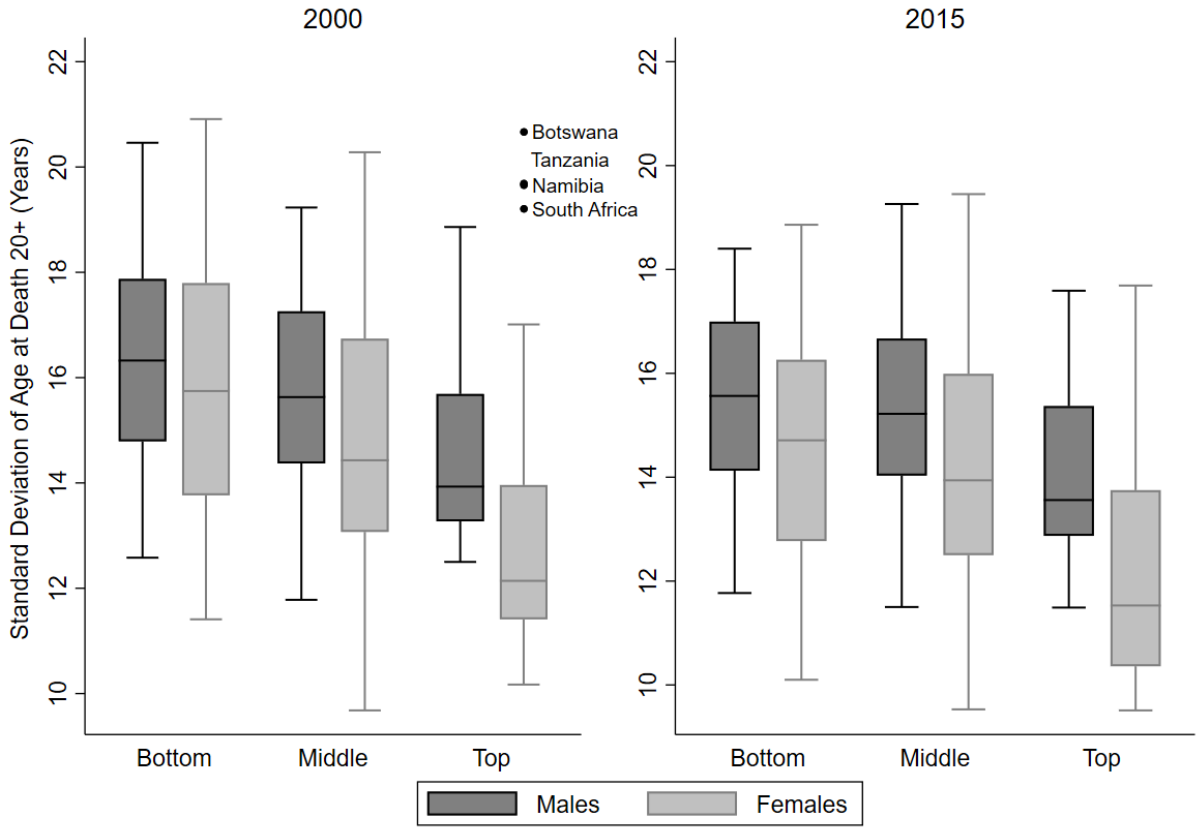
We collected annual abridged life tables for 162 countries over the 2000-2015 period from the World Health Organization's Global Health Observatory. Our dataset includes nearly all countries across the full range of economic development. We use as our dependent variables the standard deviation of age at death for ages 20 and older (S_{20}), separately for females and males, calculated with the fraction of people dying within a 5-year age group (from a standardized population of 100,000 individuals) in a given year. For illustration, see Table A1 in the Online Appendix showing Iran's life-tables for 2000 and 2015.

The standard deviation measurement is regularly used in demographic and public health research. Demographers Wilmoth and Horiuchi (1999) state the standard deviation of age at

death is one of the few measures of the variability in life tables that is easy to interpret, because it is expressed in years of age units.

To help visualize our dependent variable, Figure 3 displays the distribution of S_{20} by terciles of countries' levels of democratization for males and females for the first and last year of the period of analysis. Although males across all terciles have a higher median S_{20} than females, females have larger variation. The changes in female S_{20} from 2000 to 2015 in Botswana, South Africa, Namibia, and Tanzania (the outliers noted in the 2000 top tercile) exemplify the faster reduction of inequality in females' age at death experienced by countries in the top tercile.

Figure 3: Standard Deviation of Age-at-death by Terciles of Liberal Component Index



Inter-quartile ranges remain relatively stable for males and females across terciles of democratization, with a small increase for top-tercile females in 2015 as outlier nations in 2000

were incorporated to the overall distribution by 2015. Slight differences are detected in the upper and lower whiskers, with a notable contraction for males and females in the bottom tercile. Overall, distributions of S_{20} shifted downwards from 2000 to 2015.

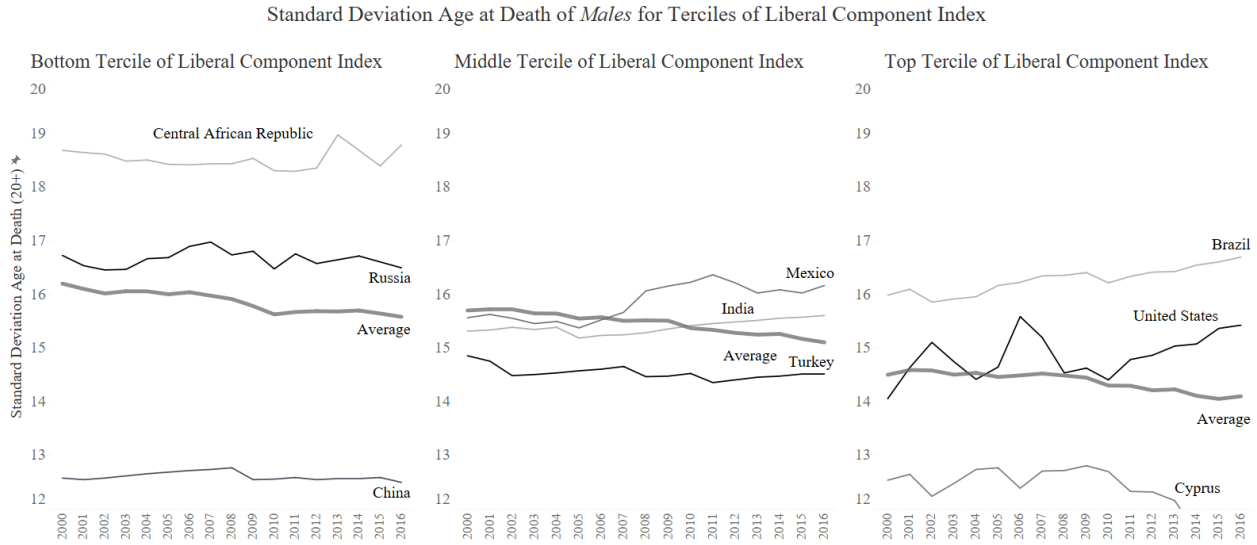
Independent Variables

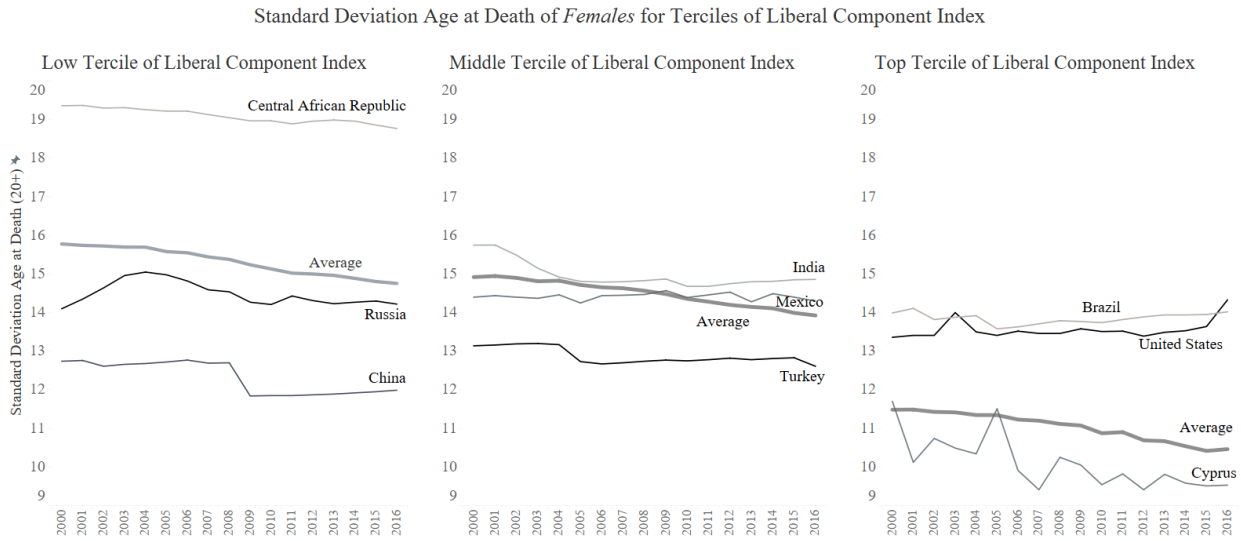
Our independent variable of interest is the Varieties of Democracy Project's (V-Dem) liberal component index (Coppedge et al. 2023). The V-Dem dataset has also been used in public health research to study the health effects of political regimes (Bollyky et al. 2019). The liberal component index gauges the democratic quality of a political regime by how much the government is constrained from taking unilateral actions against individuals and institutions. It emphasizes the rule of law and protection of individual and minority rights, especially free assembly and free press, as well as constitutional limits on executive power.

The liberal component index is an aggregate measure of the following indices: 1) equality before the law and individual liberties; 2) judicial constraints on the executive; and 3) legislative constraints on the executive (Coppedge et al. 2023). The first component of the index focuses on transparent and impartial laws and administration, and access to justice and freedom. The second component measures the judiciary's ability to act independently and the executive's compliance with court rulings. The third component measures the legislature's ability to investigate, question, and overrule the executive.[i] The index ranges from 0 to 1, with 0 indicating the total absence and 1 indicating the total achievement of liberal democratic principles. It should be noted that the meaningfulness of elections, electoral participation of citizens in the political process, and political responsiveness of the elected officials are beyond the scope of the liberal component index. Nonetheless, these factors are likely to be highly correlated with the liberal components index.

For our study period, the liberal component index has an average of 0.62, with Eritrea showing the lowest score at 0.01 and Norway the highest at 0.98. Figure 4 depicts patterns of S_{20} over time for a selection of countries at each tercile of the liberal components index, by sex. Across all terciles of the liberal components index distribution, we see overall reductions in S_{20}, with notable recent exceptions of countries in the Americas (e.g., Brazil, Mexico, United States). Perhaps the main pattern in Figure 4 is the increasingly lower S_{20} for males and females as we move from the bottom to the upper tercile of the liberal component index distribution, with overall S_{20} values markedly lower at the upper tercile.

Figure 4. Standard Deviation of Age at Death, by Sex and Regime Type





In addition to the liberal component index, we use a measure of electoral participation to capture the extent of political engagement. The variable is the total percentage of voters who cast a vote in an election (Vanhanen 2019). All other control variables come from the Quality of Government (QoG) dataset, compiled from multiple original sources (Teorell et al. 2021). We include the level of economic development, proxied by GDP per capita in 2010 US dollars from the World Development Indicators (WDI) (World Bank N.D.), and life expectancy at birth by sex (World Health Organization 2015). As discussed above, improvements in health outcomes are strongly associated with increased economic development and regime type.

Life expectancy is a crucial factor to include in our analysis, given that longer life expectancy is associated with lower premature mortality, higher economic development, and regime type. Life expectancy at birth and the standard deviation of age at death are both dimensions of mortality, and life expectancy is a standard control in epidemiological studies of S_{20} to control for aspects related to the central tendency of longevity (Wilmoth and Horiuchi 1999; Tuljapurkar 2010; Vaupel, Zhang and van Raalte 2011).

We also include standard sociodemographic controls for health outcomes in cross-national studies: unemployment rate measured as the percentage of unemployed individuals in the labor force) (ILO 2020); the percentage of rural population (World Bank N.D.), and number of years of educational attainment for individuals between 35 and 44 years of age (IHME 2015). An additional control included in our models is a one-year lagged version of the dependent variable, which should capture variation not explained by other control variables, and which offers a highly conservative estimate of the impact of our independent variables of interest.

Table 1 reports summary statistics for our analytic sample. Although the average S_{20} for females is one year less than for males (Male = 15.2 vs. Female = 14.1), its range is larger (as low as 9.4 in Kuwait and as high as 21.1 in South Africa) relative to a range of 11.2-19.4 years for males. In most countries S_{20} is lower for women than men, but in some atypical cases in Sub-Saharan Africa, such as Lesotho and Gabon, S_{20} is higher for women than men. This likely reflects persistently high maternal mortality and health outcomes associated with poverty as a main cause of high variability of women's age at death.

Table 1. Summary Statistics

Variable	Mean	Std. dev.	Min	Max
Male SD Age-at-death 20+				
Full Sample (N=2,394)	15.22	1.90	11.22	19.41
2000 (N=162)	15.46	1.96	11.78	20.46
2015 (N=162)	14.85	1.88	11.49	19.26
Female SD Age-at-death 20+				
Full Sample	14.13	2.70	9.40	21.10
2000	14.55	2.76	9.68	20.91
2015	13.56	2.48	9.51	19.45
Liberal Component Index				
Full Sample	0.62	0.27	0.01	0.98
2000	0.60	0.28	0.03	0.98
2015	0.64	0.26	0.07	0.97
Electoral Participation				
Full Sample	36.40	17.70	0.00	70.00
2000	33.50	18.98	0.00	70.00
2015	39.00	16.93	0.00	70.00
Unemployment, male (% of male labor force)				

Full Sample	7.17	5.55	0.06	36.96
2000	7.65	5.69	0.42	30.50
2015	6.96	5.33	0.06	26.72
Unemployment, female (% of female labor force)				
Full Sample	8.97	7.26	0.22	41.80
2000	9.26	7.47	0.33	43.27
2015	8.83	7.11	0.42	30.65
Rural population (% of total population)				
Full Sample	43.89	22.54	0.00	91.54
2000	46.50	22.95	0.00	91.75
2015	41.44	22.20	0.00	87.92
Educational Attainment (35-44 years, Male)				
Full Sample	9.00	3.24	1.38	15.32
2000	7.99	3.12	1.34	13.87
2015	9.91	3.25	2.02	15.32
Educational Attainment (35-44 years, Female)				
Full Sample	8.03	4.18	0.47	15.59
2000	6.92	3.92	0.44	13.96
2015	9.02	4.33	0.74	15.59
Logged GDP per capita (2010 US$)				
Full Sample	8.42	1.51	5.27	11.63
2000	8.24	1.54	5.29	11.45
2015	8.58	1.47	5.43	11.59
Life Expectancy, Male				
Full Sample	66.84	8.89	33.20	81.20
2000	63.86	9.87	38.50	77.8
2015	69.35	7.66	50.10	80.9
Life Expectancy, Female				
Full Sample	71.52	9.97	40.00	87.00
2000	68.65	11.04	40.7	84.6
2015	74.02	8.39	52.00	87.00

We also find a wide range of variation across our control variables. Given the inclusion of dictatorships in the analytic sample, the electoral participation variable ranges from 0 to 70. Although average participation increased by almost six percentage points from 2000 to 2015 (from 33.5% to 39%), the variance of this measure remained high ($SD_{2000} \approx 19$ and $SD_{2015} \approx 17$), reflecting wide differences between regime types. The overall average unemployment rate for females is larger than it is for males (8.97 and 7.17, respectively). Both distributions—for males and females—are skewed to the right with a set of countries showing particularly high levels of unemployment, mostly in Eastern Europe and Africa. Similarly, overall average educational attainment is one school-year higher for young adult males (~9 years) than for females (~8

years), with the latter exhibiting higher variation (SD_{males}=4.18 vs. $SD_{females}$=3.24). Logged GDP per capita illustrates the significant income inequality across nations, with a lowest value of 5.27 (or about $195 U.S. dollars per capita in a year) to a maximum value of 11.63 (or about $112,500 U.S. dollars per capita in a year). Lastly, life expectancy in our sample shows that females (71.5 years) tend to live longer than men (66.8 years), on average, with great inequalities, ranging between 33.2 and 81.2 years for men, and a 40 to 87 years for women, in our study period.

Methods

A central challenge of research using political variables to predict public health outcomes is the potential endogeneity of the analyzed variables. Notably, democracy has long been argued to be a function of level of development, and health outcomes themselves may in turn impact levels of development and democracy. As people have better health outcomes, they can contribute to the economy more productively and for a longer period of their life, thus improving the economy. As health improves, people have more time, accessibility, and resources to engage in politics, thus improving democratic processes as well. Accordingly, an analysis such as ours requires consideration of endogenous processes within our econometric framework.

Our estimation strategy is based on the system generalized method of moments (SGMM) estimator for dynamic models of panel data developed by Arellano and Bover (1995) and Blundell and Bond (1998). The properties of the SGMM estimator fit the purpose of our study as it addresses data limitations and supports our underlying assumptions. First, our panel structure has a large number of panels (n=162) relative to its number of periods (T=16). SGMM is appropriate for this panel structure. Second, as a first approximation and to simplify our interpretation of parameter estimates, we can assume that the underlying associations specified in our models follow a linear function. Our parameters, therefore, can be interpreted as changes in

our dependent variable with respect to a given explanatory variable independent of the value of the explanatory variable, and our parameters can also be assumed not to be a function of other parameters. We tested, yet did not find, non-linearity among variables and parameters in our models.

Third, the SGMM estimator allows our dependent variable—the standard deviation of the age-at-death distribution conditional on survival to age 20—to manifest inertia in time and behave as a country-specific slow-moving series. The SGMM is particularly appropriate for such panel dynamics. Fourth, the SGMM estimator accounts for the fact that the variables on the right side of our econometric equation (democratic liberalization and socio-economic controls) are not strictly exogenous, exhibiting inertia in time and contemporaneous correlation in their errors. This feature helps us manage endogeneity specifically. Fifth, these errors can also be assumed heteroskedastic and autocorrelated within countries, but not across them (for which we included time fixed-effects in our model specifications, removing global time-related shocks from the errors). And finally, the SGMM estimator also accounts for unobserved heterogeneity across countries.

The econometric specification of our main model is of the form:

$$\sigma_{20,it} = \alpha + \gamma\sigma_{20,i(t-1)} + \beta L_{it} + \sum_{j=1}^{n} \delta_j X_{it} + T + \varepsilon_{it} \qquad (1)$$

where $\sigma_{20,it}$ is our dependent variable—i.e., the standard deviation of age-at-death for ages 20-85+ for country *i* in time *t*. The first term $\sigma_{20,i(t-1)}$ is a 1-year lagged version of our dependent variable. L_{it} stands for the democratic liberalization component index, for country *i* in time *t*. The expression $\sum_{j=1}^{n} \delta_j X_{it}$ is a vector of *n* endogenous covariates X_{it} (in our case, the gender-specific unemployment rate, share of the rural population, logged GDP per capita, gender-

specific educational attainment in middle ages 35-44 years, voter turnout, and gender-specific life expectancy) for country *i* in time *t*, each covariate with its respective coefficient δ_j. The term *T* is a vector of time fixed effects, and ε_{it} is the error term—assumed to be independent for each country *i* for all *t*. We include the complete econometric specification of our system of equations with detailed descriptions of our SGMM instruments in the Online Appendix, Section 2, as well as tests of the underlying assumptions behind and appropriateness of our models in Section 3.

Results

The SGMM parameter estimates for six models are included in Table 2. Models 1 and 4 report the estimated effect of the liberal component index, without the inclusion of controls, on male and female S_{20}, respectively; models 3 and 6 include all controls. Results for models with only controls (i.e., excluding the liberal component index) are included in models 2 and 5.

Table 2 – SGMM Parameter Estimates, Regime Type and S_{20} by Sex

	(1) Male S_{20}	(2) Male S_{20}	(3) Male S_{20}	(4) Female S_{20}	(5) Female S_{20}	(6) Female S_{20}
Lagged Male S_{20} (t-1)	0.399***	0.487***	0.560***			
	(0.131)	(0.095)	(0.113)			
Lagged Female S_{20} (t-1)				1.057***	0.744***	0.688***
				(0.131)	(0.081)	(0.094)
Liberal Component Index	-1.661***		-1.247***	-0.095		-0.587
	(0.511)		(0.473)	(0.554)		(0.413)
Logged GDP per capita		-0.429***	-0.226		-0.163	-0.148
		(0.147)	(0.142)		(0.146)	(0.169)
Life Expectancy, Male		-0.0424**	-0.038*			
		(0.019)	(0.020)			
Life Expectancy, Female					-0.050***	-0.062***
					(0.013)	(0.018)
Electoral participation		-0.005**	0.000		-0.001	0.000
		(0.002)	(0.003)		(0.002)	(0.002)
Unemployment, Male		-0.014	-0.002			
		(0.012)	(0.011)			

Unemployment, Female					0.017	0.015
					(0.011)	(0.013)
Rural population		-0.029***	-0.020*		-0.004	-0.006
		(0.010)	(0.011)		(0.007)	(0.008)
Educational Attainment (35-44 years, Male)		-0.061	-0.062			
		(0.044)	(0.049)			
Educational Attainment (35-44 years, Female)					0.013	0.012
					(0.0247)	(0.032)
Time Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2,394	2,394	2,394	2,394	2,394	2,394
Number of countries	162	162	162	162	162	162

Note: Robust standard errors in parentheses. Statistical significance code: *** p<0.01, ** p<0.05, * p<0.1.

Our main result is that liberal democracy shows different levels of association with premature mortality by sex. The liberal component index is negative and statistically significantly related to inequality in premature mortality for males. This is not the case for females, which is still negative, yet half the size of the estimate for males and not precisely estimated.

The effect size is also substantive for men. A one-unit increase in the liberal component index (e.g., going from a country with total absence of, to one with total achievement of liberal democratic components) is associated with a decrease in male S_{20} of 1.25 years (p<0.01), on average. The size of this annual estimate is meaningful, especially when considering that the total reduction of the mean male S_{20} in our 162-country sample between 2000 and 2015 is 0.61 years (Table 1). Correspondingly, these 0.61 years are achieved by an increase in the liberal component index of 0.49 units. For instance, should a country undergo a democratic transition, such as going from the level of Nicaragua (=0.43) to that of Uruguay (=0.92), or from Myanmar (=0.15) to Thailand (=0.62), we would expect a decrease in premature mortality of 0.61 years—or, the equivalent to the decrease in premature mortality in a 15-year period. This effect is robust to model specification (including additional controls), estimation strategy, and the use of

different GMM estimators, and alternative calculations of the dependent variables. See Online Appendix Section 4.

We also consider whether economic development helps to explain reductions in premature mortality. The coefficient for logged GDP per capita is significant in the model for men without the liberal component index (Model 2); yet, once the liberal component index is included, the size of the economic effect is almost halved and it loses statistical significance (Model 3). Unlike the scenario for males, the liberal component index does not affect the coefficient size of logged GDP per capita for women, which is much smaller than for males and not statistically significant (models 5 and 6). This suggests that, if economic development has mitigating effects on female lifespan variation, then characteristics unique to liberal democratic regimes do not account for these effects, and *vice versa*.

The electoral participation measure does not have a statistically significant effect on premature mortality for either men or women when the liberal component index is included in the model. The sign of this coefficient, nonetheless, goes from positive ($\hat{\delta} = .0004$) to negative ($\hat{\delta} = -.005$) when the liberal index is excluded from the model for males, then showing a statistically significant negative effect on S_{20} (Model 2). This result may reflect a crucial importance of liberalization, including constraints on the executive and civil liberties, in reducing premature mortality of adult men, beyond the legal ability to vote. The coefficient of the electoral participation variable for females are small and do not reach statistical significance. This suggests that neither electoral nor liberal democratic mechanisms tested in our analyses appear to improve inequalities in premature mortality for women at the same level as they do for men. In our Online Appendix Table A8 we also include results with an indicator of civil society participation as alternative independent variable capturing a possible mechanism of liberal

democratic processes. Our results are consistent with the civil society participation variable substituted in our models.

Not surprisingly, we find that a higher life expectancy is associated with a decrease in S_{20}. This relationship is particularly strong for females. As their life expectancy at birth improves, so does their premature mortality. These annual estimates are also large, especially considering the slow movement of the average age-at-death, variation in the age-at-death distribution, and life expectancy in populations, and that these figures apply to the total population of 162 countries.

The estimates for the autoregressive terms show that, compared to the male $S_{20,}$ the variability in female age at death tends to remain relatively stable over time. Given the strictness of our model focusing on endogenous processes and including lag dependent variables on the right side of equation 1, we do not observe strong relationships between our controls and the dependent variables in most models. Estimates for all other sociodemographic controls either do not reach statistical significance or are substantively trivial.

Models with Causes of Death

We also test whether increased democracy reduces premature mortality for men (and women) via the potential mechanism of improved safety and reduced risk, and whether increased democracy might reduce premature mortality for women (and men) via communicable and nutritional diseases, and maternal mortality. We collected data on estimated causes of death, by gender, from the Institute for Health Metrics and Evaluation (2015). Table 3 includes parameter estimates of our base models (Models 3 and 6 in Table 2) including three relevant causes of death: fatal injuries (expected to affect men disproportionately), communicable and nutritional

diseases (expected to afflict both men and women), and maternal mortality (affecting only women). For each model, we include the sex-specific death rate according to each cause.

Model 1 in Table 3 shows support for the idea that higher deaths from injuries (from all causes of injury) are associated with higher S_{20} in men. The coefficient of the liberal component indicator remains significant in this model, but the coefficient size decreases, possibly indicating that some of the variation in premature mortality originally explained by the liberal component index could be attributed to the mortality rate due to injuries in men—with lower injury mortality rates in liberal democracies. In contrast, we do not see a significant impact of communicable disease rates on men's inequalities in premature death in Model 2. These findings are consistent with the idea that liberal democracy may improve health inequalities for men via improvements in safety that reduce risk.

We observe a reduction in the size of the coefficient of the liberal democratization indicator once female injury death rates are included in the model, suggesting the possibility that some of the association originally detected between S_{20} and liberal democratization may be partially mediated by injury death rates (Model 3). Similar to men, lower injury mortality rates

Table 3: SGMM Parameters, Including Injuries and Communicable Diseases

	(Reference Model) Male S_{20}	(1) Male S_{20}	(2) Male S_{20}	(Reference Model) Female S_{20}	(3) Female S_{20}	(4) Female S_{20}	(5) Female S_{20}
Lagged Male/Female S_{20} (t-1)	0.553***	0.721***	0.542***	0.679***	0.796***	0.664***	0.546***
	(0.112)	(0.118)	(0.112)	(0.097)	(.0513)	(0.096)	(0.103)
Liberal Component Index	-1.237**	-0.785**	-1.366***	-0.571	-0.306	-0.651	0.041
	(0.482)	(0.386)	(0.457)	(0.413)	(0.307)	(0.415)	(0.538)
Logged Male/Female Injuries Rate		0.571* (0.296)			0.247 (0.204)		
Logged Male/Female Communicable and			-0.027 (0.384)			0.108 (0.121)	

Nutritional Diseases Death Rate							
Logged Maternal Mortality Rate							0.840*** (0.190)
Time Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Control Variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2,378	2,378	2,378	2,378	2,378	2,378	2,378
Number of countries	161	161	161	161	161	161	161
F(b) Full Model	0.17	0.25	0.21	0.22	0.25	0.23	0.21

for women are observed in liberal democracies. Both coefficients for women, nevertheless, remain non-statistically significant. In contrast, we observe an increase in the size of the liberal democratization coefficient once communicable disease death rates are included in the model (Model 4). Although both coefficients remain non-statistically significant, this finding suggests that the association between female's premature mortality and liberal democratization may be partially mediated by higher communicable disease death rates for women in liberal democracies than in non-liberal ones.

As expected, we find that women's S_{20} is highly related to maternal mortality (Model 5). We also find that, once the maternal mortality rate is included in the model, its coefficient is large and statistically significant while the coefficient for liberal democratization collapses toward zero (Model 5). This finding suggests that, even though the coefficient for liberal democratization was not precisely estimated, maternal mortality mediates nearly the totality of its impact. The negative (although statistically insignificant) association between premature mortality of females and liberal democratization in the reference models appears to be explained by differences in maternal mortality rates between liberal and non-liberal democracies—with liberal democracies producing lower maternal mortality rates than non-liberal ones. Our estimates are consistent with the idea that improvements in life expectancy and the standard

deviation of age at death for women come in great part from reductions in maternal mortality for young and young-adult women of child-bearing age.

Discussion and Conclusion

Our results provide empirical support for the proposition that democratic liberalization improves premature mortality. We find a robust, large, and statistically significant association between the degree of liberal democracy and the contraction of inequalities in premature mortality across nations. The effect, although favorable in both instances, is only precisely estimated for males and not for females. Our findings are empirically reliable and causally informative as we account for endogenous processes related to health outcomes, democracy, economic development, electoral participation, and life expectancy, among others. Our findings suggest that liberal democratization may generate a feedback-loop of political advantages: as nations experience lower premature mortality, their citizens have more opportunities to participate in politics and increase their community involvement, which in turn produce a more informed and active citizenry—all fundamental conditions for healthy and stable democracies. We also discuss several possible mechanisms that may help to explain the reduction in adult male's premature mortality associated with liberal democracy, including less exposure to occupational and unintentional injuries or road accidents in comparison to less liberal regimes. As nations move up the ladder in democratic principles, they tend to invest more in the sectors that improve occupational safety and decrease accidents and injuries. These types of policies disproportionately reduce premature death inequalities among men.

We also find evidence, although weaker, suggesting that inequalities in female premature mortality may be improved by liberal democratization. Even though our liberalization index coefficients are not precisely estimated, we do find evidence suggesting that its association with

premature mortality among females is mediated through the improvement of maternal conditions. The stability of the models, the strength of the associations, and the precision of estimates favor the greater benefits that males can achieve from the advancement of democratic principles, as opposed to females. These results emphasize the paradox that democratic principles, while likely enhancing the quality of life for citizens, may not necessarily address gender equality.

Our research may be particularly relevant to public health as we are experiencing a period of democratic backsliding around the world and a recent decline in life expectancy in the United States (Bermeo 2016, Mechkova, Lührmann, and Lindberg 2017, Waldner and Lust 2018). If democracy encourages politicians to improve health and safety, or if it provides opportunities for citizens to engage in collective action to improve health outcomes, we may see stagnation in global gains in gender health equality and in the reduction of premature mortality among men. Son and Bellinger (2022) find empirical evidence of the high cost of authoritarianism to public health. They show that a rise in authoritarianism reduces healthcare spending, increases infant mortality, and decreases female life expectancy. The authors posit that weakening of liberal democratic components of democracy, such as freedom of expression, association, clean elections, and undermining of legislature by the executive are all associated with adverse health outcomes. While global trends in health outcomes have been showing success for several decades, this progress may not continue at the same pace as many nations limit political freedoms. At the same time, paradoxically, gender inequalities in premature death may decrease, not because of the stagnation of health improvements for females due to the deterioration of health improvements for males.

Our research findings have implications regarding the potential for greater liberalization to influence health policies even in non-democracies. Though citizens of non-democracies may have limited agency to challenge the state directly, it may be possible to influence autocratic decision makers through informal networks and the pressures of media. Access to information related to health issues could be expanded by strengthening grassroots advocacy and independence of media. Additionally, people in non-democracies may be able to work directly with key constituencies that comprise a base of support for the regime and influence policy changes that will improve public health. These efforts may be particularly efficacious to improve outcomes if influential social groups can stress to autocratic governments potential improvements in economic productivity among working age people, and how reductions in premature mortality may help the economy.

Research into political processes within authoritarian regimes shows ostensibly democratic institutions, such as political parties and elections, however stage-managed they might be by autocratic rulers, are important structures to garner support from key constituencies and offer democratic validity for authoritarian control (Ghandi 2008). Most autocratic nations have some "popular" institutions, including voting for legislatures or local government officials. Such political opportunities may be beneficial for citizens in non-democracies to demand improvements in health through adoption of laws and regulations and allocation of resources to minimize risks of premature death from childbearing, occupational hazards, and poorly maintained infrastructure.

Our study has also implications for international multilateral organizations and non-profits focused on public health. These groups may improve public health outcomes by explicitly recognizing the association between democracy, including the role of civil society and social

movement pressures, and health outcomes. Our research suggests it is not only the affluence and material resources of democracies that positively affect people's health, but the degree of citizen involvement and input into decision-making. Institutional oversight over the executive, rule of law, and civil liberties and rights may be key for health improvements. The political empowerment of citizens likely needs to come from within the society and its political values, but these organizations can provide opportunities for education and resources to push for improvements for both men and women.

Our research findings also put into focus the importance for these organizations to adopt strategies for equal access to vaccinations and medication to tackle communicable diseases, especially among young and middle-aged adults. Financial aid and loans could also include programming to improve regulations related to workplace hazards and more aggressive improvements in sanitation, hygiene, and infrastructure.

Our research considers variation in premature adult mortality across the globe and provides supporting evidence that political liberalization may be one of the possible means toward effective action for global health. The most pressing needs for mitigation are in places with very high adult premature mortality; for one critical example, certain Sub-Saharan African nations known for high prevalence of HIV and maternal mortality. Latin America is also an important region to examine as variation in premature mortality has increased due to interpersonal violence, road injuries, and ischemic heart diseases as the leading causes (Wang et al. 2016). The Americas is also the region of the world in which levels and inequality in premature mortality are growing worse, including the United States (Bound et al. 2015, 2018; Geronimus et al. 2019). El Salvador is a case in point, with extremely high inequalities in male

lifespans. The trends in the Americas suggest that for middle- and high-income countries, addressing causes of preventable death may be critical.

There are several limitations of this study. First, our analysis includes a large sample of 162 countries, representing a wide range of variation in economic and government systems across the globe. Yet, our results may not be generalizable to especially small and poor nations, which were excluded from our study due to lack of data availability. Second, our liberal component index is a crude, overall estimate that attempts to capture as much variation as possible about factors that previous research linked to processes of liberal democracy. Although our chosen index is well-established in the literature, and available for a large sample of countries, the process that generates the variable as well as each of its underlying components may induce non-essential variation and measurement error in the overall index indicator. As such, our findings should be interpreted in the context of summary indexes of liberal democracy and not in the context of specific characteristics of regimes. Future research should examine the specific mechanisms whereby democracies reduce premature mortality, and on how to make those favorable effects spill over to females. We have theorized certain mechanisms, especially risk reductions in the workplace, on the road, and due to violent conflict, that could be tested with more detail in subsequent studies.

The implications of our research for global health are such that achieving equity in global outcomes via international cooperation and aid is more likely if the sources of these inequities lie in economics, medical technological advancement and knowledge, or education. Global health inequalities driven by politics are likely to only be addressed by social and political development at the national and local levels, which may be improved by increased liberalization.

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Does Democracy Matter for Lifespan Inequalities? Regime Type and Premature Mortality by Sex

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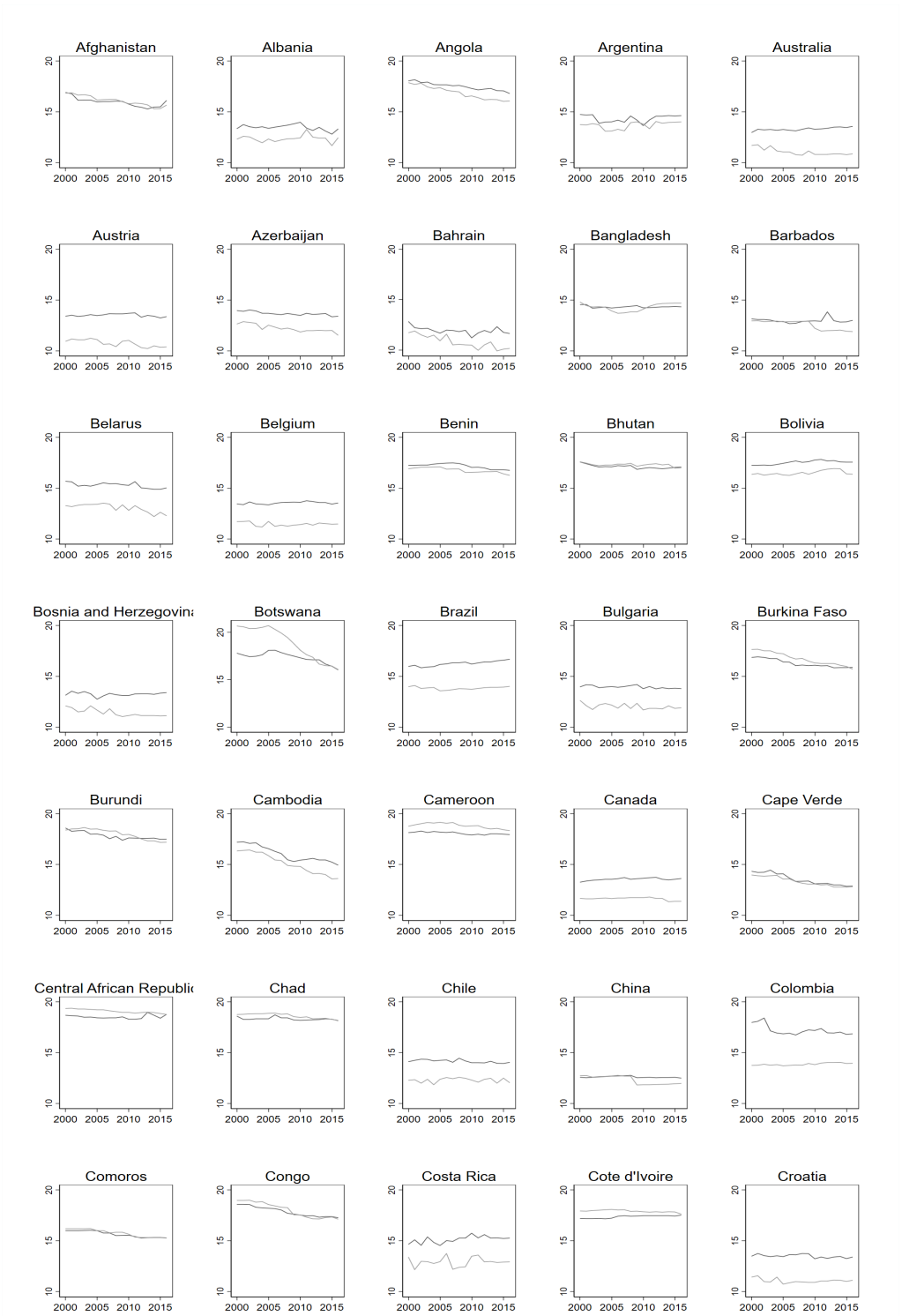
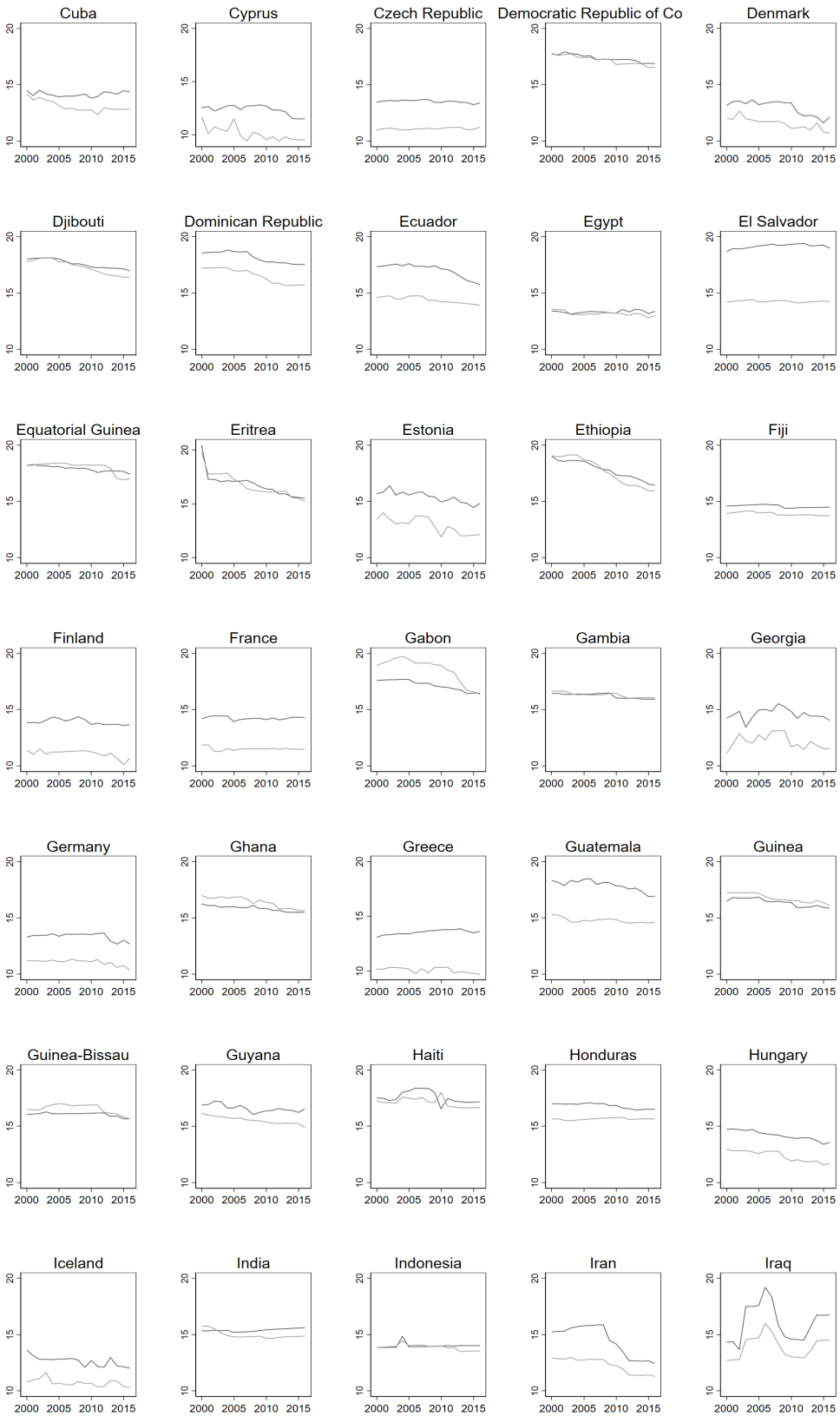
Section 1: Summary Data

Figure A1 – Standard Deviation Age at Death Over Time for Males and Females by Country

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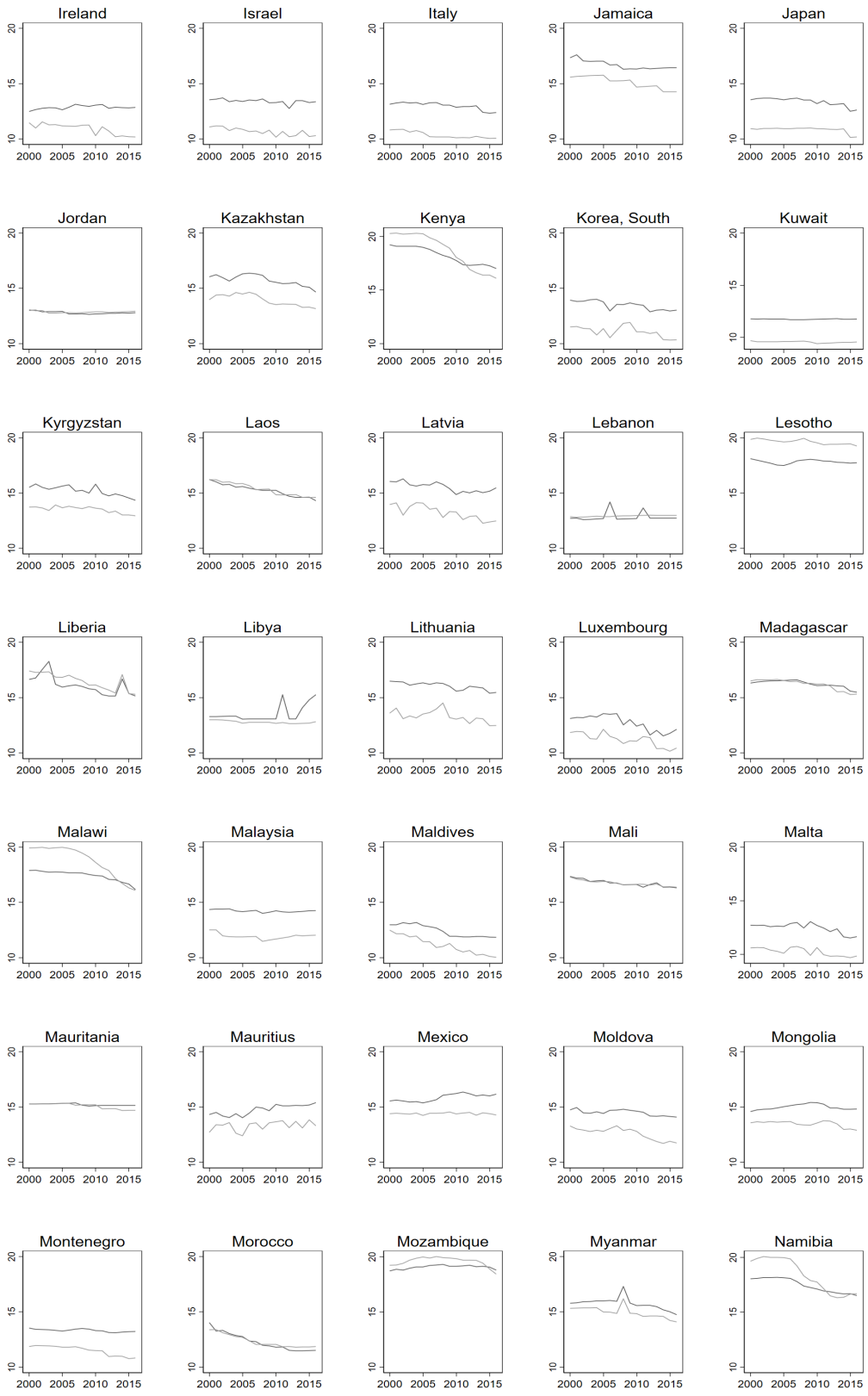
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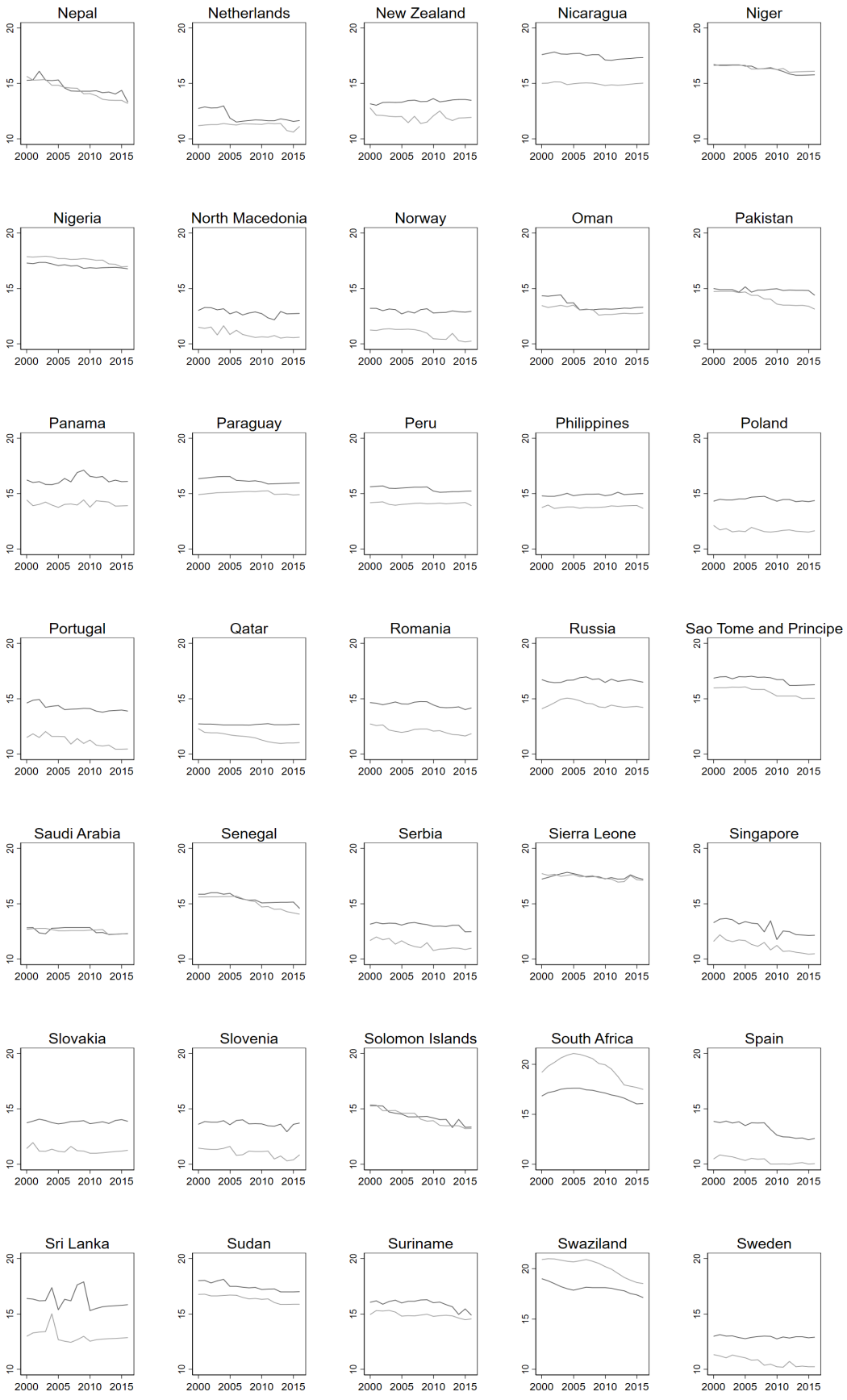
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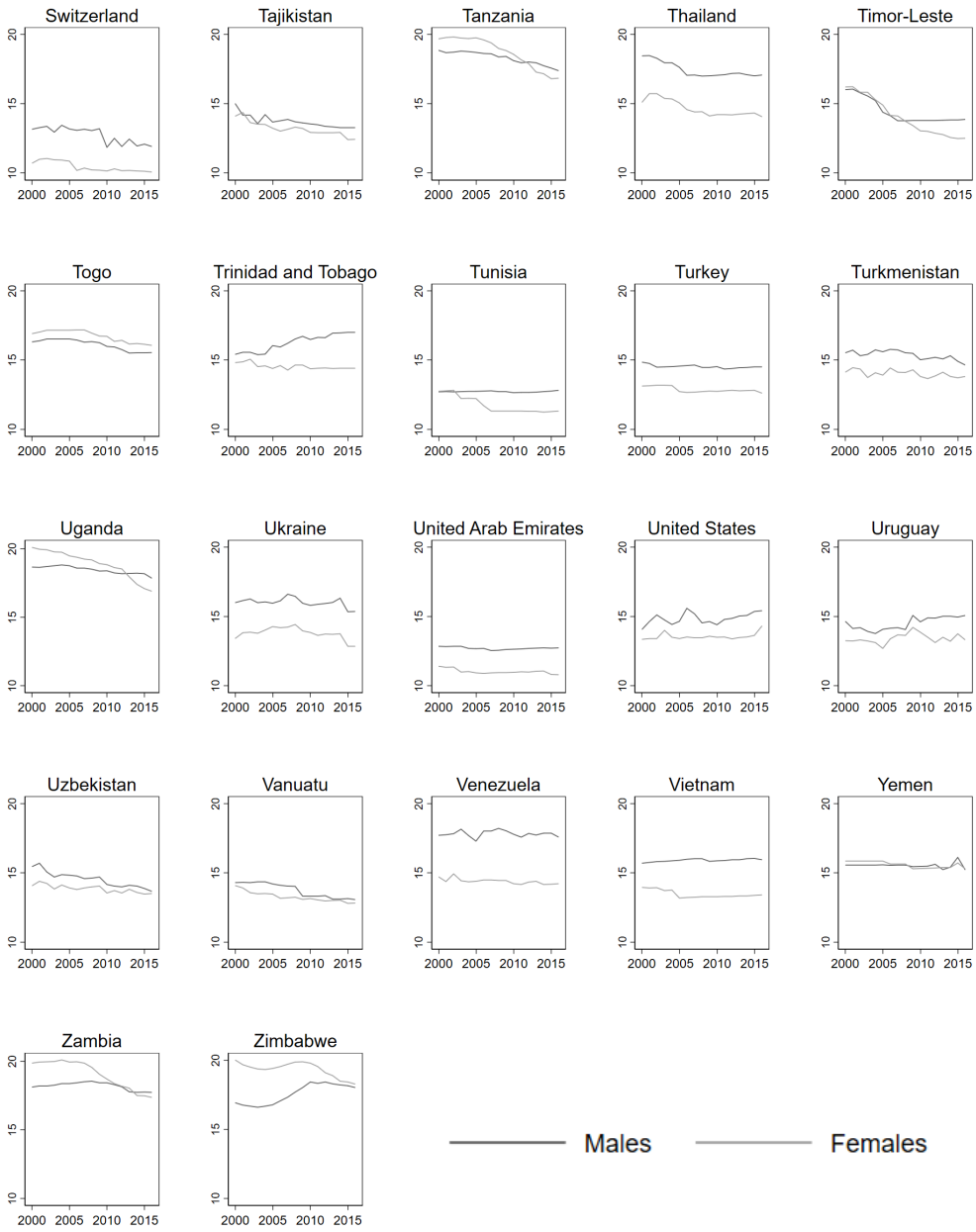
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Section 2: Additional Description of the SGMM Estimator and Our Method

The SGMM estimator exploits all available information by combining all sets of moment conditions from the differenced and level equations—explicitly, by instrumenting differenced variables in the model with their own lagged levels, and by conversely instrumenting lagged variables with their differenced conditions, all for each available period. This augmented version of the Arellano-Bond GMM estimator goes beyond within or between first-difference methods by taking advantage of both across-time and between-countries variation. This SGMM specification also leads to considerable improvements in the asymptotic properties of the estimator, unbiasedness, and precision (Blundell and Bond 1998).

Originally, Arellano and Bond (1991) used the work of Anderson and Hsiao (1982) and Holtz-Eakin, Newey, and Rosen (1988) on autoregressions with panel data to introduce a first-difference GMM estimator that corrects for the endogenous behavior of the independent variables and for the correlation between the errors and lags of the dependent variable in linear dynamic panel models. Specifically, when the dependent variable is a function of its past values (i.e., a lagged version of the dependent variable is included as a regressor), the OLS fixed effects estimator will produce a biased estimate of the autoregressive parameter. This is because the within transformation of the two-way fixed effects model (i.e., subtracting the individual's mean value of each variable from itself) creates a correlation between the lagged dependent variable and the error. Furthermore, if other regressors are correlated with the lagged dependent variable, their coefficients will also be biased. One way in which Arellano and Bond's first-difference GMM estimator solves these problems is by using lagged levels of the dependent and independent variables (from the second most recent period up to all preceding periods) as instruments for the first-differenced variables in the model.

Yet Arellano and Bover (1995) note that more moment conditions are available for identification beyond those used by the first-difference GMM estimator. Also, Blundell and Bond (1998) show that first-difference GMM estimators would produce imprecise estimates and may not be particularly informative since, in many instances, the lagged versions of the variables are weak instruments for the differenced variables in the model. If this is so, then the first-difference estimator would be particularly biased when the levels of the independent variables are persistent in time—as often occurs with political variables in comparative analyses.

As stated in the manuscript, this is our main econometric model specification:

$$\sigma_{20,it} = \alpha + \gamma\sigma_{20,i(t-1)} + \beta L_{it} + \sum_{j=1}^{n} \delta_j X_{it} + T + \varepsilon_{it} \qquad (1)$$

For instruments selection, we use the Sargan-Hansen and difference-in Hansen tests as a guide to avoid an excessively large set of instruments that could overfit our endogenous variables. By sequentially testing incremental lag specifications (e.g., first with a 2-year lag, then with 2- and 3-year lags, and so on), both tests rejected the validity of the overidentifying restrictions—e.g., when using 2- to 5-year lags for the first-differenced equation (Eq. 2 below). As such, we use 2- to 4-year lags in our models. The tests also indicate that only the first differences are valid as

instruments for the level equation (Eq. 3 below) after using 2- to 4-year lags in the first-differenced equation as instruments (see Figure A2 and Tables A2-A3).

The set of moment conditions in which lagged levels are used as instruments for the differenced version of Eq. 1, and lagged differences are used as instruments for the level version of Eq. 1—both instances specified, separately, for each variable in the right side of Eq. 1—can be expressed as, respectively:

$$\Delta Z_{it} = \varphi + \sum_{l=2}^{4} \theta_l Z_{il} + \mu_{it} \qquad (2)$$

where ΔZ_{it} stands for the first-differenced version of any variable Z_{it} in the right side of Eq. 1 (namely $\sigma_{20,i(t-1)}$, L_{it}, and all X_{it}), such that $\Delta Z_{it} = Z_{it} - Z_{i(t-1)}$. The term $\sum_{l=2}^{4} \theta_l Z_{il}$ is a vector of l lagged versions of the specific Z_{it} (in our case, from the 2nd up to the 4th lag of Z_{it}) each with its respective coefficient θ_l; and μ_{it} is the error term. Likewise,

$$Z_{it} = \omega + \rho \Delta Z_{it} + \tau_{it} \qquad (3)$$

where Z_{it} stands for the level version of any variable in the right side of Eq. 1 (namely $\sigma_{20,i(t-1)}$, L_{it}, all X_{it}, and T). Accordingly, the term $\Delta Z_{it} = Z_{it} - Z_{i(t-1)}$ (while for $\sigma_{20,i(t-1)}$, $\Delta\sigma_{20,i(t-1)} = \sigma_{20,i(t-1)} - \sigma_{20,i(t-2)}$), where only first-difference versions of the specific Z_{it} are used. τ_{it} is the error term. The system of equations above (comprised by equations 1 through 3) was fitted separately for males and females.

Other methodological considerations are worth mentioning. Given that the number of differenced and lagged instruments can grow large relative to our sample size, our SGMM approach could manifest issues of instrument proliferation, thus overfitting our endogenous variables and therefore biasing and reducing precision of parameter estimates (Beck, Levine and Loayza 2000; Roodman 2009). We thus followed Arellano (2003) to reduce overparameterization and capped the count of instruments by truncating the lag depth. Under this approach, the number of instruments per period is constrained to the most-immediate (i.e., the most relevant) lags (Soto 2009; Roodman 2009). It is expected that, after a certain number of lagged periods, their coefficients would become uninformative about endogeneity—i.e., the coefficient estimates of further lags would become undifferentiable from "0." To keep the number of instruments under the number of cases, we implemented lagged instruments up to 3 periods (e.g., in Eq. 2, l allows for 2nd, 3rd, and 4th lags of the levels of all endogenous covariates; see Figure A2).

To further avoid instrument proliferation, we also followed Roodman (2009) and "collapsed" the instrument set, such that a set of instruments for each available lag is generated (as opposed to a set of instruments for each lag *and* period). Although statistical ways to determine the adequate count of instruments are still underdeveloped, Windmeijer (2005) reports that for a two-step GMM estimator, reducing the number of instruments by about half reduced bias by about 40%. Reducing overidentification also has been shown to reduce linearity in T in model specifications like Eq. 1 above (Vieira et al. 2013).

Considering that the consistency of the SGMM estimator depends on the validity of the instruments, we checked for possible over-identification of the restrictions using a series of Sargan-Hansen and difference-in-Hansen tests (tables A2-A3) (Sargan 1958; Hansen 1982; Roodman 2009). We also used the Arellano-Bond test (Arellano and Bond 1991) to test the null hypothesis of no serial error correlation (Table A4). We did not assume strict exogeneity in any instance; all our variables in the right side of Eq. 1 were considered endogenous. To avoid downward bias in our standard errors, we used the two-step robust estimation developed in Windmeijer (2005). We additionally report results from one-step and iterated SGMM (Hansen et al. 1996) in Table A5. Results show that the liberal component index coefficient estimate is robust to the use of different GMM estimators.

Section 3: Tests of SGMM Model Assumptions

Figure A2: Liberal component index estimate as the number of instruments increased

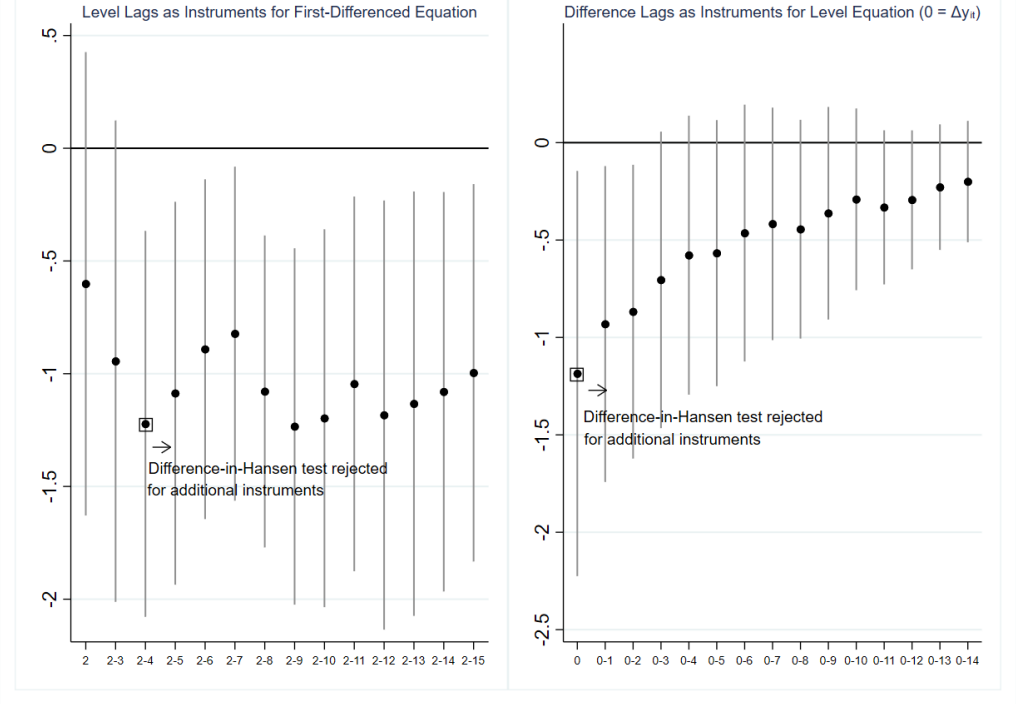

Table A1: Life Table - Iran 2000 and 2015

(Highlighted columns depict the underlying data of the age-at-death distributions illustrated in Figure 1 in the main text)

Age Group	(nMx) age-specific death rate between ages x and x+n		(nqx) probability of dying between ages x and x+n		(lx) number of people left alive at age x		**(ndx) number of people dying between ages x and x+n**		(nLx) person-years lived between ages x and x+n		(Tx) person-years lived above age x		(ex) expectation of life at age x	
2000														
	Male	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
≤1 year	0.029	0.027	0.028	0.027	100000.0	100000.0	2801.4	2659.5	97559	97690	6908655	7117872	69.1	71.2
1-4 years	0.002	0.002	0.006	0.007	97198.6	97340.5	588.4	649.9	387360	387724	6811096	7020181	70.1	72.1
5-9 years	0.001	0.001	0.003	0.004	96610.2	96690.6	317.7	341.0	482257	482601	6423735	6632457	66.5	68.6
10-14 years	0	0	0.002	0.002	96292.5	96349.7	211.3	196.0	480934	481258	5941478	6149857	61.7	63.8
15-19 years	0.002	0.001	0.007	0.005	96081.2	96153.6	719.8	484.2	478814	479637	5460544	5668599	56.8	59
20-24 years	0.003	0.001	0.013	0.006	95361.4	95669.4	1195.7	617.9	473936	476831	4981730	5188962	52.2	54.2
25-29 years	0.002	0.001	0.012	0.006	94165.7	95051.5	1159.3	604.7	467930	473753	4507794	4712131	47.9	49.6
30-34 years	0.003	0.001	0.013	0.007	93006.4	94446.8	1186.1	651.0	462090	470646	4039864	4238378	43.4	44.9
35-39 years	0.003	0.002	0.014	0.009	91820.3	93795.8	1267.7	812.6	456024	467031	3577774	3767731	39	40.2
40-44 years	0.004	0.002	0.018	0.012	90552.6	92983.2	1664.2	1070.8	448800	462394	3121750	3300700	34.5	35.5
45-49 years	0.005	0.004	0.025	0.018	88888.4	91912.5	2246.7	1633.6	439157	455782	2672950	2838306	30.1	30.9
50-54 years	0.008	0.006	0.039	0.029	86641.8	90278.8	3406.0	2623.7	425137	445297	2233793	2382524	25.8	26.4
55-59 years	0.01	0.009	0.051	0.043	83235.8	87655.1	4217.0	3799.9	406122	429467	1808656	1937228	21.7	22.1
60-64 years	0.015	0.015	0.075	0.074	79018.8	83855.3	5892.7	6219.1	381207	404930	1402535	1507761	17.7	18
65-69 years	0.028	0.027	0.133	0.128	73126.1	77636.1	9754.8	9911.0	343192	365206	1021327	1102831	14	14.2
70-74 years	0.052	0.048	0.233	0.215	63371.3	67725.1	14750.9	14572.2	282174	304372	678136	737625.1	10.7	10.9
75-79 years	0.084	0.088	0.347	0.359	48620.4	53152.9	16857.5	19103.0	201233	218295	395961	433253.5	8.1	8.2
80-84 years	0.129	0.122	0.484	0.463	31762.9	34049.9	15376.2	15748.4	119021	129416	194729	214958.7	6.1	6.3
85+ years	0.216	0.214	1	1	16386.7	18301.5	16386.7	18301.5	75708	85542	75708	85542.27	4.6	4.7
2015														
	Male	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
≤ 1 year	0.029	0.027	0.028	0.027	100000.0	100000.0	2801.4	2659.5	97559	97690	6908655	7117872	69.1	71.2
1-4 years	0.002	0.002	0.006	0.007	97198.6	97340.5	588.4	649.9	387360	387724	6811096	7020181	70.1	72.1
5-9 years	0.001	0.001	0.003	0.004	96610.2	96690.6	317.7	341.0	482257	482601	6423735	6632457	66.5	68.6
10-14 years	0	0	0.002	0.002	96292.5	96349.7	211.3	196.0	480934	481258	5941478	6149857	61.7	63.8
15-19 years	0.002	0.001	0.007	0.005	96081.2	96153.6	719.8	484.2	478814	479637	5460544	5668599	56.8	59
20-24 years	0.003	0.001	0.013	0.006	95361.4	95669.4	1195.7	617.9	473936	476831	4981730	5188962	52.2	54.2
25-29 years	0.002	0.001	0.012	0.006	94165.7	95051.5	1159.3	604.7	467930	473753	4507794	4712131	47.9	49.6
30-34 years	0.003	0.001	0.013	0.007	93006.4	94446.8	1186.1	651.0	462090	470646	4039864	4238378	43.4	44.9
35-39 years	0.003	0.002	0.014	0.009	91820.3	93795.8	1267.7	812.6	456024	467031	3577774	3767731	39	40.2
40-44 years	0.004	0.002	0.018	0.012	90552.6	92983.2	1664.2	1070.8	448800	462394	3121750	3300700	34.5	35.5
45-49 years	0.005	0.004	0.025	0.018	88888.4	91912.5	2246.7	1633.6	439157	455782	2672950	2838306	30.1	30.9
50-54 years	0.008	0.006	0.039	0.029	86641.8	90278.8	3406.0	2623.7	425137	445297	2233793	2382524	25.8	26.4
55-59 years	0.01	0.009	0.051	0.043	83235.8	87655.1	4217.0	3799.9	406122	429467	1808656	1937228	21.7	22.1
60-64 years	0.015	0.015	0.075	0.074	79018.8	83855.3	5892.7	6219.1	381207	404930	1402535	1507761	17.7	18
65-69 years	0.028	0.027	0.133	0.128	73126.1	77636.1	9754.8	9911.0	343192	365206	1021327	1102831	14	14.2
70-74 years	0.052	0.048	0.233	0.215	63371.3	67725.1	14750.9	14572.2	282174	304372	678136	737625.1	10.7	10.9
75-79 years	0.084	0.088	0.347	0.359	48620.4	53152.9	16857.5	19103.0	201233	218295	395961	433253.5	8.1	8.2
80-84 years	0.129	0.122	0.484	0.463	31762.9	34049.9	15376.2	15748.4	119021	129416	194729	214958.7	6.1	6.3
85+ years	0.216	0.214	1	1	16386.7	18301.5	16386.7	18301.5	75708	85542	75708	85542.27	4.6	4.7

Table A2: Sargan-Hansen (difference) test of the overidentifying restrictions (Difference Equation)

		Excluding			Difference		
Moment conditions		Chi2	Df	p-value	Chi2	df	p-value
Lag 2	First-Differenced Model (Autoregressive Term)	10.6232	6	0.1007	0.0038	1	0.9505
	First-Differenced Model (Covariates)	0.0034	1	0.9533	10.6236	6	0.1007
	Level Model	.	-14	.	.	.	.
	First-Difference Model	0.0104	0	.	10.6166	7	0.1562
Lags 2 and 3	First-Differenced Model (Autoregressive Term)	15.3652	12	0.2221	0.9982	2	0.6071
	First-Differenced Model (Covariates)	0.4476	2	0.7995	15.9158	12	0.1951
	Level Model	.	-7	.	.	.	.
	First-Difference Model	0	0	.	16.3634	14	0.2917
Lags 2 to 4	First-Differenced Model (Autoregressive Term)	24.7317	18	0.1325	1.428	3	0.699
	First-Differenced Model (Covariates)	2.1824	3	0.5354	23.9772	18	0.1558
	Level Model	0	0	.	26.1596	21	0.2004
	First-Difference Model	0	0	.	26.1596	21	0.2004
Lags 2 to 5	First-Differenced Model (Autoregressive Term)	31.2402	24	0.147	6.3928	4	0.1717
	First-Differenced Model (Covariates)	3.2555	4	0.516	34.3775	24	**0.0782**
	Level Model	7.4227	7	0.3862	30.2103	21	**0.0878**
	First-Difference Model	0.0031	0	.	37.6299	28	0.1056

Table A3: Sargan-Hansen (difference) test of the overidentifying restrictions (Level Equation)

		Excluding			Difference		
Moment conditions		Chi2	Df	p-value	Chi2	df	p-value
First Difference	First-Differenced Model (Autoregressive Term)	24.732	18	0.133	1.428	3	0.699
	First-Differenced Model (Covariates)	2.182	3	0.535	23.9772	18	0.1558
	Level Model	0	0	.	26.1596	21	0.2004
	First-Difference Model	0	0	.	26.1596	21	0.2004
First Difference and First Lag of Difference	First-Differenced Model (Autoregressive Term)	49.316	25	**0.003**	0.7567	3	0.8598
	First-Differenced Model (Covariates)	27.790	10	**0.002**	22.283	18	0.2197
	Level Model	0	0	.	50.0727	28	**0.0063**
	First-Difference Model	11.218	7	0.129	38.8551	21	**0.0102**

Table A4: Arellano-Bond test for autocorrelation of the first-differenced residuals

	Z	p-value
Order 1	.	.
Order 2	0.1995	0.8419

Section 4: Robustness Checks

In this section we include models to assess the robustness of our inferences. In Table A5 we show our results are similar when using One-Step and Iterated SGMM approaches.

Table A5: One-Step and Iterated SGMM (Males)

	Male SD Age at Death 20+ (One-Step)	Male SD Age at Death 20+ (Iterated)
Male SD Age at Death 20+ (t-1)	0.573***	0.560***
	(0.130)	(0.133)
Liberal Component Index	-1.116**	-1.282**
	(0.553)	(0.545)
Participation		0.000
		(0.003)
Unemployment, male (% of male labor force)	-0.007	-0.001
	(0.015)	(0.011)
Rural population (% of total population)	-0.012	-0.022
	(0.013)	(0.014)
Educational Attainment (35-44 years, Male)	-0.055	-0.060
	(0.047)	(0.068)
Logged GDP per capita (constant 2010 US$)	-0.062	-0.265
	(0.232)	(0.170)
Life Expectancy, Male	-0.045	-0.038
	(0.029)	(0.026)
Constant	11.84**	13.81***
	(4.590)	(4.89)
Observations	2,394	2,394
Number of countries	162	162

Note: Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

The empirical specification of our main model (Eq. 1) includes economic variables (i.e., GDP per capita, unemployment rate, and rurality) that should, in theory, remove from our coefficient of interest variation related to the provision of health services and other relevant economic determinants of adult premature mortality. Still, to test the robustness of our findings, we additionally run models that include indicators of government health expenditure (Table A6) and globalization (Table A7), using variables obtained from the World Development Indicators.

The inclusion of government health expenditure per capita does not affect the size or statistical significance of the liberal component index. Its coefficient does not reach statistical significance, which is expected due to the presence of other highly correlated regressors such as GDP per capita. We also include trade openness (calculated as the sum of imports and exports as a percentage of GDP) as a measure of globalization. Although we lose five countries due to data missingness, the coefficient of interest only decreases by 5% when this control is added to the model.

Table A6: Results with Government Health Expenditure per capita (Males)

	(1) Male S_{20}	(2) Male S_{20}
Lagged male S_{20} (t-1)	0.556***	0.570***
	(0.115)	(0.117)
Liberal Component Index	-1.275**	-1.262**
	(0.502)	(0.501)
Logged Gov. Health Exp. Per cap		*0.0530*
		(0.194)
Unemployment, male	-0.002	-0.001
	(0.011)	(0.010)
Electoral participation	0.001	0.001
	(0.003)	(0.002)
Rural population	-0.020*	-0.018
	(0.012)	(0.012)
Educational Attainment (35-44 years, Male)	-0.0636	-0.070
	(0.048)	(0.048)
Logged GDP per capita	-0.216	-0.251
	(0.138)	(0.277)
Life Expectancy, Male	-0.040*	-0.039*
	(0.021)	(0.021)
Constant	13.51***	13.20***
	(4.081)	(4.292)
Time Fixed Effects	Yes	Yes
Observations	2,391	2,391
Number of countries	162	162

Note: Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

Table A7: Results with Indicator of Globalization (Males)

	(1) Male S_{20}	(2) Male S_{20}
Lagged male S_{20} (t-1)	0.544***	0.530***
	(0.113)	(0.124)
Liberal Component Index	-1.149**	-1.212***
	(0.470)	(0.396)
Unemployment, male	-0.001	0.002
	(0.011)	(0.011)
Electoral participation	0.001	0.001
	(0.003)	(0.003)
Rural population	-0.019	-0.023*
	(0.012)	(0.012)
Educational Attainment (35-44 years, Male)	-0.0743	-0.0607
	(0.048)	(0.046)
Trade Openness (exp + imp % of GDP)		*-0.002*
		(0.002)
Logged GDP per capita	-0.204	-0.238
	(0.145)	(0.157)
Life Expectancy, Male	-0.042**	-0.048**
	(0.020)	(0.021)
Constant	13.64***	14.76***
	(4.017)	(4.532)
Observations	2,279	2,279
Number of countries	157	157

Note: Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

We also theorized that collective action specifically aimed at affecting policy to minimize premature mortality risks is a key potential mechanism through which liberal democratization can reduce the standard deviation of age at death. To explore this claim, we include V-dem's measure of civil society participation as a measure of collective action (Table A8). This indicator captures whether civil society "enjoys autonomy from the state and in which citizens freely and actively pursue their political and civil goals however conceived" (Coppedge et al., p.51, 2023). The liberal component and the civil society participation indices are highly correlated, both conceptually and statistically, with a correlation coefficient of 0.82. We did not include them in the same model due to high multicollinearity. Both indices are produced by the V-dem project, and so are calculated as aggregations of data evaluated by overlapping country-experts.

We find, in Models 1 and 4, that the civil society participation index shows a similar relationship to S_{20} as the liberal component index. The civil society variable, which captures one of our

hypothesizes mechanisms, is a reasonable alternative measure of liberal democratic processes. Thus, we find support for the idea that the civil society engagement facets of liberal democracy are associated with reduced morality inequality in men, but not significantly in women (even if the coefficient value is negative).

As an effort to disentangle the effect of each of these measures on adult premature mortality, we experimented with a residualized version of the dependent variable (Ridker and Henning 1967). Specifically, we removed the variation of the standard deviation of age at death that can be individually explained by the civil society participation index through a bivariate regression, from which we recover the residuals. We then use these residuals (i.e., everything about S_{20} that cannot be explained by the civil society participation index) as the dependent variable to test whether the liberal component index can explain variation in S_{20} beyond that already explained by the civil society participation index. We repeat the same process, residualizing the dependent variable on the liberal component index, to test whether the civil society participation index can explain variation in S_{20} beyond that already explained by the liberalization index.

Models 2 and 3 in Table A8 show the results of these tests for men: While the effect of the liberal component index remains statistically and substantively significant, the civil society participation index is not able to explain the residualized variation in S_{20}. These results suggest that between the civil society participation and the liberalization indices, the liberalization index is the one carrying the underlying essential variation that explains male premature mortality (i.e., the explanatory power of the civil society participation index may be attributed to its correlation with the liberalization index, and not to its unique association with premature mortality). Even though the two processes (democratic liberalization and civil society participation) are intrinsically related, it is democratic liberalization process that lies beneath the variation in the social determinants of health that ultimately influence premature mortality. Models 5 and 6 show the results for women. Consistent with previous results, the liberal component index coefficient has a smaller negative effect on the residualized variation in female $S_{20,}$ but it does not reach statistical significance. Lastly, civil society participation is again not able to explain the residualized variation in female S_{20}.

Table A8: Results with Civil Society Indicator and Residualized Dependent Variable (Males)

	(1) Male S_{20}	(2) Male S_{20} Residualized on Civil Society	(3) Male S_{20} Residualized on Liberal Democracy	(4) Female S_{20}	(5) Female S_{20} Residualized on Civil Society	(6) Female S_{20} Residualized on Liberal Democracy
Lagged S_{20} (t-1)	0.556***	0.568***	0.557***	0.683***	0.698***	0.652***
	(0.109)	(0.108)	(0.087)	(0.093)	(0.087)	(0.092)
Liberal Component Index		**-0.899***			**-0.124**	
		(0.460)			**(0.350)**	
Civil Society Participation	**-1.208***		**-0.235**	**-0.803**		**0.088**
	(0.511)		**(0.409)**	**(0.557)**		**(0.567)**
Unemployment, sex-specific	-0.008	-0.005	-0.004	0.009	0.015	0.018
	(0.010)	(0.011)	(0.010)	(0.015)	(0.013)	(0.015)
Electoral participation	0.002	0.000	0.002	0.000	0.001	0.004
	(0.003)	(0.002)	(0.003)	(0.002)	(0.002)	(0.003)
Rural population	-0.014	-0.018	-0.022**	-0.003	-0.004	-0.003
	(0.011)	(0.020)	(0.010)	(0.009)	(0.007)	(0.010)
Educational Attainment (35-44 years, sex-specific)	-0.073	-0.056	-0.068	0.003	0.008	0.012
	(0.052)	(0.047)	(0.042)	(0.039)	(0.032)	(0.043)
Logged GDP per capita	-0.166	-0.227	-0.230*	-0.139	-0.129	-0.205
	(0.147)	(0.144)	(0.121)	(0.183)	(0.162)	(0.188)
Life Expectancy, sex-specific	-0.036*	-0.035*	-0.042**	-0.062***	-0.061***	-0.052**
	(0.018)	(0.021)	(0.020)	(0.017)	(0.018)	(0.021)
Constant	12.20***	6.139**	6.389***	10.02***	5.428***	5.109**
	(3.843)	(2.430)	(1.908)	(3.354)	(1.989)	(2.346)
Time Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2,372	2,372	2,372	2,372	2,372	2,372
Number of countries	160	160	160	160	160	160

Note: Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Including individuals over 84 can potentially generate instability in the standard deviation of the distribution. This is mostly because death certificates information for oldest older adults tends to be more unreliable across nations. Table A9 indicates that the size of the estimated effect of the liberal component index does not change much (from the one reported in the manuscript ($\hat{\beta} = -0.587$)) for female S_{20} when the 85 and older age group is excluded from the calculation of the dependent variable. The coefficient is now, nevertheless, statistically significant at the 0.1 level. The size of the effect changes a bit more for male S_{20} (from the one reported in the manuscript ($\hat{\beta} = -1.247$)), while remaining statistically significant at the 0.05 level, suggesting that oldest older men may be particular beneficiaries of democratic liberalization.

Table A9: Results with Standard Deviation Age at Death (20 – 84) (Males)

	(2) Male S_{20}	(4) Female S_{20}
Lagged male S_{20} (t-1)	0.643***	
	(0.131)	
Lagged Female S_{20} (t-1)		0.684***
		(0.075)
Liberal Component Index	-0.926**	-0.613*
	(0.453)	(0.348)
Logged GDP per capita	-0.103	-0.055
	(0.144)	(0.114)
Life Expectancy, Male	-0.040**	
	(0.018)	
Life Expectancy, Female		-0.068***
		(0.019)
Electoral participation	0.001	-0.001
	(0.002)	(0.002)
Unemployment, male	0.001	
	(0.009)	
Unemployment, female		0.007
		(0.010)
Rural population	-0.013	-0.007
	(0.011)	(0.007)
Educational Attainment (35-44 years, Male)	-0.074*	
	(0.045)	
Educational Attainment (35-44 years, Female)		-0.009
		(0.023)
Constant	10.30**	10.19***
	(4.361)	(2.800)
Time Fixed Effects	Yes	Yes
Observations	2,393	2,393
Number of countries	162	162

Note: Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

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Endnotes

[i] Empirical tests show that measures of electoral aspects of democracy operationalized in more fined-grained levels and aggregated in a multiplicative manner are more likely to show an impact on mortality outcomes, such as the infant mortality rate (Gerring et al. 2021). V-Dem's liberal component index is additively aggregated as an average of the aforementioned components, which can make a statistically significant association less likely.