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RESEARCH ARTICLE



Public health, public protest: The role of health burdens and healthcare access in protest mobilisation

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ABSTRACT

Health and politics are intertwined, yet few studies have examined the association between health and protest. This study examined whether population health burdens were associated with protest incidence and whether healthcare access modified these associations. Analysis was based on an unbalanced 2004–2023 country-year panel, combining protest counts from ACLED with rates for 22 GBD causes. Mixed-effects negative-binomial models estimated incidence-rate ratios (IRRs) with interactions for healthcare access (± 1 SD). Two-way fixed-effects Poisson models were estimated as a benchmark to distinguish cross-national associations from within-country dynamics. Health burdens were systematically, but heterogeneously, associated with protest. Rates for several non-communicable burdens were associated with protest, notably musculoskeletal disorders (IRR 1.72, 95% CI 1.37–2.15), neoplasms (1.24, 1.06–1.44), substance-use disorders (1.32, 1.12–1.56) and HIV/AIDS and other STIs (1.24, 1.12–1.38). Higher healthcare access generally attenuated health–protest associations. Fixed-effects models confirmed several associations (e.g. HIV/AIDS, neoplasms) but revealed that others (e.g. maternal/neonatal disorders, enteric infections) were driven primarily by cross-national differences. Population health burdens were associated with cross-national variation in protest mobilisation. Chronic, non-communicable burdens were associated with heightened protest, whereas poverty-linked and early-life burdens were associated with lower mobilisation. Healthcare access was associated with attenuation of these relationships.

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Introduction

Politics shapes health in a range of ways. Political decisions determine who has access to care, how resources and opportunity are distributed and the groups or populations that are prioritised or overlooked. More generally, politics has substantial influence on the social, economic and environmental conditions that underpin health. Health also shapes politics in a range of ways. Health outcomes often drive political priorities and agendas, with health disparities offering a reflection of broader inequality and neglect. While there has been a substantial and growing body of evidence that has explored the impact of politics on health (Bollyky et al., 2019), fewer studies have examined the impact of health on politics. One notable exception has been the empirical evidence on health and political engagement, which has described how health, whether good, bad or otherwise shapes engagement with politics, in a range of ways (Mattila et al., 2017; Michener, 2018). The mechanisms that underpin health and political engagement can be understood through what has been labelled the health-gap/politicisation hypothesis (Anderson et al., 2022). This hypothesis synthesises resource and grievance-based theories and suggests that poor/good health can both promote and constrain political engagement. That is, poor health promotes political engagement as it makes grievances more likely, at the same time it may suppress engagement as those with poor health have less capacity or resources to engage with politics. Similarly, the inverse is true for good health, in that it may promote engagement, as those with

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Table 1. Health-gap/politicisation hypothesis (adapted from Anderson et al., 2022)

		Political engagement	
		Low	High
Health	Good	(Reverse) politicisation hypothesis	Health gap hypothesis
	Bad	Health gap hypothesis	Politicisation hypothesis

good health have greater resources or capacity to do so, at the same time those with good health may also have fewer grievances owing to their health status which may act to suppress political engagement. This model is summarised in Table 1.

The fact that poor/good health can both promote and suppress political engagement is reflected in the empirical literature, with results across several studies supporting this divergence. On one hand, there is evidence to suggest that better health is associated with greater political efficacy (Shore et al., 2019). Conversely, studies have also shown that poorer health is associated with less political engagement and satisfaction with political systems. For example, amongst a sample from several European countries, those who had poorer health were generally less engaged with politics and less satisfied with political systems (Anderson et al., 2022). Similar findings have come from the US, with those who reported poor health less likely to vote (Pacheco & Fletcher, 2015). At the same time however, evidence suggests that grievances may also play a role, with political engagement motivated by poor health. In a US study, it was found that after adjusting for a range of socioeconomic and health-related factors, individuals with cancer diagnoses were more likely to vote, while those with heart disease diagnoses were less likely to do so (Gollust & Rahn, 2015). Other studies suggest that it may also be the form of political action and/or engagement that matters. While throughout Europe it was found that increased levels of disability meant that people were less likely to vote, it increased other forms of political engagement, such as contacting politicians and participating in demonstrations, something which was also driven by perceived discrimination (Mattila & Papageorgiou, 2017). Similar findings were reported from Denmark, Finland, Iceland, Norway and Sweden where people with poor health were more active than others in making direct contact with power holders and participating in demonstrations (Söderlund 201538; Rapeli,). Other studies suggest a similar pattern, notably that how we measure health matters when we speak about political engagement, whether we are speaking about self-reported health or being limited in daily activities, the type of health burden¹ we speak about can influence political engagement in different ways (Burden et al., 2017; Stockemer & Rapp, 2019). While the empirical evidence is largely consistent with the health-gap/politicisation hypothesis, these studies suggest that matters are more complicated. Notably, the poor/good health binary hardly captures the range of experiences people have in relation to their health or how it may impact capacity. At the same time, political engagement also comes in a range of forms, as we saw in the above studies, from voting to lobbying to contacting politicians and government, and is shaped by institutional and political contexts that are dynamic and shift over time. While the evidence seems to suggest that it may be a combination of things, from the type of health burden we are speaking about to the more specific form of political engagement, we are a long way from understanding the reasons why health may promote or suppress engagement under different circumstances and for people facing different health burdens.

Protest² is a varied but specific type of political activity and sits as distinct from many other forms of political action, with marches, sit-ins and other forms of public (often collective) action contrasting to more contained, institutionalised forms of political engagement, like voting and lobbying.³ In parallel to the above literature that has sought to explain the impact of health on political engagement, a literature that has sought to explain mobilisation in relation to social movements and protest has been developed over several decades. We find overlap, with the work above and with theories that have focused on mobilisation of social movements and protest, with explanations that account for grievances, (Power et al., 2020), but that also propose factors such as political opportunities, mobilisation structures, framing processes and protest cycles as important in explaining the mobilisation and success (or otherwise) of social movements and protest (Benford & Snow, 2000; McAdam et al., 2003; Meyer & Minkoff, 2004). While this literature has been influential and applied widely to explaining protest and social movement mobilisation, health has been largely overlooked in these discussions. This study sought to contribute to both literatures above in bringing health into conversation with protest, more specifically examining the role that health burdens and healthcare access have in explaining protest mobilisation.

Research questions

This study sought to examine (1) the extent to which population-level health burdens were associated with the incidence of protest events, after adjusting for economic, political and social controls, and whether these associations reflected within-country temporal dynamics or primarily cross-national structural differences and (2) whether access to healthcare and country income modify these associations.

Methods

To examine the above questions, a country-year panel spanning 2004–2023 was assembled from several sources. A negative binomial mixed-effects specification was utilised as preliminary diagnostics suggested protest counts exhibited substantial overdispersion. Within each model, we specified country and year level random intercepts to account for unobserved, time-invariant heterogeneity between countries and for global shocks or trends common to each year. All health burden variables and selected controls entered the model as fixed effects. The data sources utilised, data transformations undertaken and the analytic strategy utilised are detailed below. All analyses were performed in R 4.4.0 (Posit team, 2025), a full list of packages utilised is included as Supplementary material (Table S1).

Data sources

Data for this study comes from several sources. To quantify protests per country per year we utilised the Armed Conflict Event Location Database (ACLED, 2024). ACLED provides a database that tracks violence and disorder globally, including protest events. ACLED coverage started with some countries in 1997 and by 2021 all countries globally were covered. Our analysis included all ACLED years from 2004–2023, with the dataset constructed around this, that is, where a country was not covered data was treated as missing, where a country was covered but no protests were recorded, the assumption was made that the country in question had no protests that year. ACLED data coverage is summarised in Tables S3–5. The Varieties of Democracy (V-DEM) dataset was also utilised. This dataset covers 201 countries from 1789–2024 on a range of social and political variables, including measures related to democracy, civil liberties, political equality, media freedom, corruption and institutional performance (Coppedge et al., 2025). We utilised variables from this dataset to examine the social, political environment and levels of healthcare access. Institute for Health Metrics and Evaluation's (IHME) Global Burden of Disease (GBD) 2023 data was also utilised to obtain cause-specific age-standardised death rates and DALY rates, for each of the 22 'level 2' burdens (Institute for Health Metrics & Evaluation (IHME), 2025). The World Bank's World Development Indicators (WDI) were utilised for data on macro-economic (i.e. GDP, CPI), educational and demographic characteristics of countries (World Bank, 2025). All variable descriptive statistics and missingness are reported in Tables S2–S7.

A country-year panel was assembled that summarised the number of protests per country-year and other forms of violence (from the ACLED data), governance, political and civic-space covariates (from V-DEM data), macro-economic, demographic and educational characteristics by country (from WDI data) and health outcome variables (from GBD data). All variables and data sources are provided in Table S2 (Supplementary material). The descriptive statistics for variables included in the final analysis (see below) are included in Table S3 (Supplementary material). The years of coverage by country-year for each variable are included in Tables S3–7 (Supplementary material).

Data transformation

Annual protest counts were extracted from the Armed Conflict Location & Event Database (ACLED) for all countries with coverage between 2004 and 2023. As noted above, as ACLED data has not covered every country over this period, an unbalanced panel was created. If there was no coverage data was treated as missing, if there was coverage but no protests, it was assumed that no protests occurred in that country during that year. The same process was undertaken with events labelled as 'battles', while we were not primarily interested in violent action, we utilised this variable as a potential covariate in our models below, as we assumed that levels of violence may influence protest incidence. For each of these variables we utilised country-year population

to offset in models and calculate protest and rates per 100,000 population. All covariates were standardised (z), with scores calculated across the entire unbalanced panel (i.e. subtracting the global mean and dividing by the global standard deviation) rather than within countries. For DALY and death rate variables a one-year lag was also calculated. Income tertiles were computed from time-varying GDP PPP. Healthcare access (from V-DEM data) was mean-centred and scaled (± 1 SD) to ease interpretation of interaction terms.

Analytic strategy

In all models below, the outcome of interest was protest incidence by country-year. In the first models we sought to examine the association between the 22-health burdens (both DALY and death rates) and protest rates, while controlling for the most influential political, economic and demographic factors in each model. Further models examined whether healthcare access and equity and country income modified these associations, and whether country income further modulated these dynamics. All models were fitted with complete data only, omitting records with missing values and forgoing any imputation procedures. Analysis was undertaken in five steps. Each will be outlined below.

Selection of covariates

While we were primarily interested in how DALY and death burdens impacted protest incidence, there was also a need to control for the range of factors that could also influence protest incidence, from political openness, governance, demographic, economic and geographic factors. Initially, we assembled a pool of candidate variables that were theoretically grounded and drawn from existing empirical evidence. That is, GDP/economic development (including urbanisation) (Korotayev et al., 2021), inequality (Jo & Choi, 2019; Solt, 2015), economic inflation/pressures (Hendrix et al., 2009; Hossain & Hallock, 2022), violence (Krtsch, 2025) and democratic openness/opportunity (Jami & Peoples, 2022) and the interaction of several of these factors (Rød & Weidmann, 2023; Rød et al., 2025) all of which have been found to influence rates of protest. All variables that were considered are included in Table S2.

To identify which variables were the most influential and in taking a transparent approach to covariate selection, we first screened for variance-inflation factors (VIF) and data completeness; variables with more than 50% missing observations and $VIF > 10$ were examined. We then estimated a Poisson LASSO ($\alpha = 1$) regression with country-year protest counts as the outcome, population size as an exposure offset and both health and control covariates as predictors. Health variables were always forced into the model (penalty factor = 0), while controls were subject to shrinkage (penalty factor = 1). The optimal penalty parameter (λ) was chosen via 10-fold cross-validation stratified by year (~90% training, 10% validation per fold), minimising mean squared prediction error. Candidate controls with non-zero coefficients at λ min were identified as potential confounders. To further assess robustness, we performed bootstrap stability selection with 200 resamples, refitting the LASSO in each replicate and tabulating the frequency of inclusion for every control. Controls retained in at least 80% of resamples were judged stable. Guided by these analyses six variables were retained as covariates, GDP PPP, Gini Index, Consumer Price Index (CPI), Freedom of Association index, battles per 100,000 population and urbanisation (% of population who lived in urban settings). Elsewhere similar approaches have been utilised and been found to be effective in identifying true confounders and predictors of outcome while excluding irrelevant covariates (Shortreed & Ertefaie, 2017).

To ensure robustness and provide an explicit theoretical rationale for the final adjustment set, we then constructed a directed acyclic graph (DAG) specifying assumed causal relationships among health burden, protest incidence and all candidate covariates. Under the assumed causal structure (Figure S1), the DAG-implied minimal adjustment set comprised conflict, GDP, healthcare access, inequality, inflation and urbanisation. Our empirical control set matches this closely, substituting political openness (freedom of association) for healthcare access. Political openness was included on theoretical grounds and is confirmed by the canonical adjustment set. Healthcare access was deliberately excluded from the main specification because it lies on the causal pathway from structural determinants to health burden; adjusting for it would estimate only the direct effect of health burden on protest, net of access-mediated pathways. Healthcare access instead enters the analysis as an explicit interaction term to examine its moderating role. This approach estimates the total effect of health burden on protest in the main models and the access-conditional effect in the interaction models.

The final models therefore contain all cause-specific health variables, the offset for population exposure and the above subset of economic, demographic and governance controls. The results of these analyses and the variables that were assessed for eligibility are summarised in Tables S8 and 9 (Supplementary material).

Health burdens and protest incidence

The main analyses sought to answer the first research question, namely the extent to which population-level health burdens predicted the incidence of protest events, after adjusting for economic, political and social controls. In the first set of analyses, the dependent variable was the annual count of protest events. Primary exposures were cause-specific burden DALY and mortality rates. As noted above, each exposure was standardised (z) and a one-year lag was created. Covariates and their selection are listed above; each covariate was also standardised. In each specification we estimated a mixed-effects negative-binomial model, which took the form of

$$y_{it} \sim \text{NB}(\mu_{it}, \theta), \log \mu_{it} = \beta_0 + \beta_1 \text{Health}_{it} + \sum_{k=1}^K \gamma_k \text{Control}_{kit} + u_i + v_t + \log\left(\frac{\text{Pop}_{it}}{100000}\right).$$

where y_{it} was the number of protest events by country i and calendar year t ; θ was the NB2 dispersion parameter; Health_{it} was the standardised, cause-specific DALY or mortality-rate predictor (analysed both contemporaneously and with a one-year lag); and the K covariates in comprise log-GDP PPP, Gini index, CPI, freedom of association index, battles per 100k population and urban population. u_i and v_t were random intercepts capturing unobserved, time-invariant heterogeneity between countries and global shocks common to each calendar year, respectively. An offset of $\log\left(\frac{\text{Pop}_{it}}{100000}\right)$ scaled population to a per-100 000 rate. Parameters were estimated in glmmTMB (Brooks et al., 2017) with the nbinom2 family; exponentiated coefficients are reported as incidence-rate ratios (IRR).

To distinguish cross-national associations from within-country temporal dynamics, we also estimated two-way fixed-effects (FE) Poisson models for each cause, absorbing all time-invariant country heterogeneity and year-specific shocks:

$$y_{it} \sim \text{Poisson}(\mu_{it}), \log(\mu_{it}) = \beta^1 \text{Health}_{it} + \sum \gamma_k \text{Control}_{kit} + \alpha_i + \delta_t + \log(\text{Pop}_{it}/100,000).$$

where α_i and δ_t were country and year fixed effects. This specification identified associations solely from within-country temporal variation, that is, it asked whether changes in a country's health burden over time were associated with changes in its protest incidence, conditional on country-level time-invariant factors and global year effects. Coefficients from the FE specification were manually exponentiated to obtain IRRs.

To address the multiple comparisons problem arising from estimating models across 22 DALY and 21 mortality causes, we applied Benjamini–Hochberg false discovery rate (FDR) correction to all contemporaneous p -values and, separately, to all lagged p -values. We also report Bonferroni-corrected p -values as a conservative benchmark (Table S12).

Healthcare access as a moderator of protest

To test whether healthcare access modified the health-protest association, we added an interaction term between each health predictor and healthcare access and equity (from V-DEM data). This variable asks, 'to what extent is high quality basic healthcare guaranteed to all, sufficient to enable them to exercise their basic political rights as adult citizens?' (Coppedge et al., 2025) and was preferred over other measures of healthcare access and quality as it provided consistent country-year coverage over the study period and was directly related to political engagement⁴. This model took a similar form to the main model:

$$y_{it} \sim \text{NB}(\mu_{it}, \theta), \log \mu_{it} = \beta_0 + \beta_1 \text{Health}_{it} + \beta_2 \text{Access}_{it} + \beta_3 \text{Health}_{it} \times \text{Access}_{it} + \sum_{k=1}^K \gamma_k \text{Control}_{kit} + u_i + v_t + \log\left(\frac{\text{Pop}_{it}}{100000}\right).$$

From each fitted interaction model, we computed marginal IRRs for the health predictor in question at three values of access namely at -1 SD (low access), 0 SD (mean access) and $+1$ SD (high access) and we also calculated the exponentiated interaction coefficient as the per-SD change in healthcare access.

As with the main models, we estimated FE Poisson versions of the health $\times$ access interaction, replacing the random intercepts with country and year fixed effects. This allows us to assess whether the moderating role of healthcare access holds when exploiting within-country variation only, or whether it is primarily a cross-national phenomenon.

We also estimated a pooled three-way interaction (health $\times$ access $\times$ income tertile) under the RE specification to explore whether the health–protest relationship varied across national income levels. These results are reported in Supplementary material (Tables S15, Figures S2 and 3).

Model-diagnostics procedures

All above models were subjected to post-estimation checks to verify distributional assumptions and overall fit. Using the DHARMA package, we generated 1000 simulated residual draws per model and applied three omnibus tests: the dispersion test (for residual over/under-dispersion), the zero-inflation test and the Kolmogorov–Smirnov uniformity test. We computed these p -values for every (i) main-effect model, (ii) health $\times$ healthcare access interaction model and (iii) income-specific healthcare $\times$ access model.

Sensitivity and robustness checks

To assess the stability of the above findings beyond the RE–FE comparison, we implemented a battery of additional checks. These addressed three potential issues, namely, model specification, distributional assumptions and sample composition. For model specification, we estimated a series of models that systematically varied the breadth of adjustment. Beginning with bivariate models (health exposure only), we sequentially introduced domain-specific control blocks, economic (GDP-PPP, Gini, CPI), political (freedom of association), social disorder and violence (battles per 100k population) and geographic (urbanisation), before re-estimating the full model that contained all six controls (Table S17). We also replaced the two-way RE structure with a simpler random-intercept NB2 model absorbing country heterogeneity only, and fitted a zero-inflated NB2 model with an intercept-only inflation equation to assess excess zeros. For distributional assumptions, we addressed the influence of extreme protest counts by trimming the upper one per cent of the outcome distribution and re-estimating a two-way FE Poisson model on the restricted sample. We also examined temporal sensitivity by restricting the panel to 2010–2023, a period characterised by greater data completeness (Table S18). For sample composition, we compared included versus excluded country-years on key characteristics (GDP, democracy scores, urbanisation, population, protest counts) and by ACLED region (Table S19). We estimated a reduced specification excluding the Gini index, the covariate with the highest missingness (Table S20). We also estimated all main RE NB2 models on a restricted subset of countries with $\geq 80\%$ temporal ACLED coverage across the analysis window, to assess sensitivity to ACLED reporting heterogeneity (Tables S21).

Results

Descriptive overview

The complete merged dataset used for analysis spanned 2004–2023 and included 1456–1460 complete country-year rows, dependent on the health burden in question. While protest data were non-missing for 241 countries/territories (with ≥ 1 protest in 226), the final models below included protests recorded across 132 countries after excluding missing data. The analytic panel was heavily unbalanced: from 2004–2017 the sample comprised 48–77 country-years, expanding to 160 in 2018 and 243 by 2021–2023 as ACLED coverage broadened (Table S4). In terms of missing data, political, economic and demographic covariates were generally 84.9%–93.1% complete overall (political: 84.9%; demographic and conflict measures: 93.1%), while economic variables ranged from 72.3% (Gini) to 90.0% (GDP) complete, with CPI 84.2% complete. Health burden data was 90.1% complete for DALY and mortality rates, with the exception of sense-organ diseases mortality which had no coverage. Countries included in the analytic sample were substantially wealthier (median GDP PPP ~ 64 billion vs ~ 14 billion), more democratic (polyarchy 0.46 vs 0.35), more civically liberal (0.64 vs 0.49) and recorded more protests (median 43.5 vs 6.0 per country-year) than excluded country-years (Tables S19). The number (and rate) of protests increased over 2004–2023, with a skewed distribution. The top five countries accounted for 35.5% and the top ten for 48.9% of all protests. When it came to healthcare access, mean scores increased over 2004–2023 (from -0.61 to 0.42 on average; 84.9% coverage). Descriptive statistics related to the

health-burden variables used here have been reported widely elsewhere in GBD studies (e.g. Naghavi et al., 2024). Descriptive statistics and coverage for variables are included in Tables S3–S7 (Supplementary materials).

Health burdens and protest incidence

Negative-binomial models were fitted for 22 DALY and 21 mortality burdens, each estimated contemporaneously and with a one-year lag. These models indicated that population-standardised health burdens were systematically associated with protest incidence, with the direction and magnitude varying by cause and, to a lesser extent, by temporal specification. Higher DALY rates for several non-communicable and chronic conditions were associated with increased protest activity, for example, musculoskeletal disorders (IRR = 1.72, 95% CI 1.37–2.15), neoplasms (1.24, 1.06–1.44), substance use disorders (1.32, 1.12–1.56) and HIV/AIDS and other STIs (1.24, 1.12–1.38). In contrast, DALY burdens commonly linked to poverty and early-life risks were associated with fewer protests, including maternal and neonatal disorders (0.44, 0.34–0.56), nutritional deficiencies (0.14, 0.07–0.26), neglected tropical diseases and malaria (0.72, 0.61–0.84), skin and subcutaneous diseases (0.61, 0.49–0.76), transport injuries (0.70, 0.58–0.84) and enteric infections (0.79, 0.64–0.98) all associated with lower levels of protest. Several estimates were close to unity and not statistically significant (e.g. respiratory infections and tuberculosis, chronic respiratory diseases, neurological disorders, diabetes and kidney diseases, self-harm and interpersonal violence, unintentional injuries). A one-year lag calculated for both DALY and mortality rates largely preserved the contemporaneous findings, consistent with a temporal ordering in which health burdens precede protest behaviour. Results of these models are summarised in Figure 1 and in Tables S10 (Supplementary material).

Two-way FE Poisson models estimated alongside the RE specification revealed important distinctions in the source of these associations (Table S11, Figure 2). For a subset of causes, RE and FE estimates converged in direction and magnitude, providing the strongest evidence of association. HIV/AIDS showed consistent positive associations under both specifications (RE 1.24, FE 1.29 for DALYs; RE 1.23, FE 1.29 for deaths), meaning that both countries with higher HIV burden tended to have more protests, and within a given country, years with higher HIV burden were also years with more protests. Neoplasms showed a similar pattern (RE 1.24, FE 1.31 for DALYs; RE 1.37, FE 1.40 for deaths). Self-harm and interpersonal violence was consistently null-to-negative under both (~0.94).

For several poverty-linked burdens, however, the RE and FE estimates diverged substantively. Maternal and neonatal disorders showed RE = 0.44 but FE = 2.72 (wide CI); enteric infections RE = 0.79 but FE = 2.65; nutritional deficiencies RE = 0.14 but FE = 3.30 (very wide CI). This indicates that the negative RE associations for these causes reflected structural differences between countries, countries with high maternal mortality are typically poorer, less democratic and record fewer protests for reasons extending well beyond maternal health, rather than a within-country dynamic whereby worsening maternal health suppressed protest. Substance use disorders showed the opposite pattern: a positive RE (1.32) but non-significant negative FE (0.70), suggesting that this association too was primarily cross-national. FE estimates were typically less precise than RE estimates, reflecting the limited within-country temporal variation in slow-moving health burden measures.

After applying Benjamini–Hochberg FDR correction across all 43 contemporaneous models (22 DALY, 21 mortality), 12 DALY causes and 11 mortality causes remained significant at $q < 0.05$. Ten DALY and eight mortality causes survived the more conservative Bonferroni correction. The strongest associations, musculoskeletal disorders, other NCDs, maternal/neonatal disorders, nutritional deficiencies, sense organ diseases, skin diseases, NTDs/malaria, transport injuries, substance use, HIV/AIDS and neoplasms, were robust to FDR correction. Causes that did not survive FDR (e.g. enteric infections DALYs, digestive diseases deaths) had been marginal in the uncorrected analysis. Full FDR results are reported in Table S12.

Healthcare access as a moderator of protest incidence

We re-estimated every model with an explicit health-burden $\times$ healthcare-access interaction and formally tested whether the strength of the health-protest association changed per 1 SD increase in healthcare access. Overall, greater access generally attenuated the association across a wide set of causes. For DALY rates, significant protest-dampening was observed for chronic respiratory diseases (IRR per SD = 0.84, 95% CI 0.72–0.98, $p = 0.024$), transport injuries (0.80, 0.68–0.95, $p = 0.009$), digestive diseases (0.75, 0.64–0.88, $p < 0.001$),

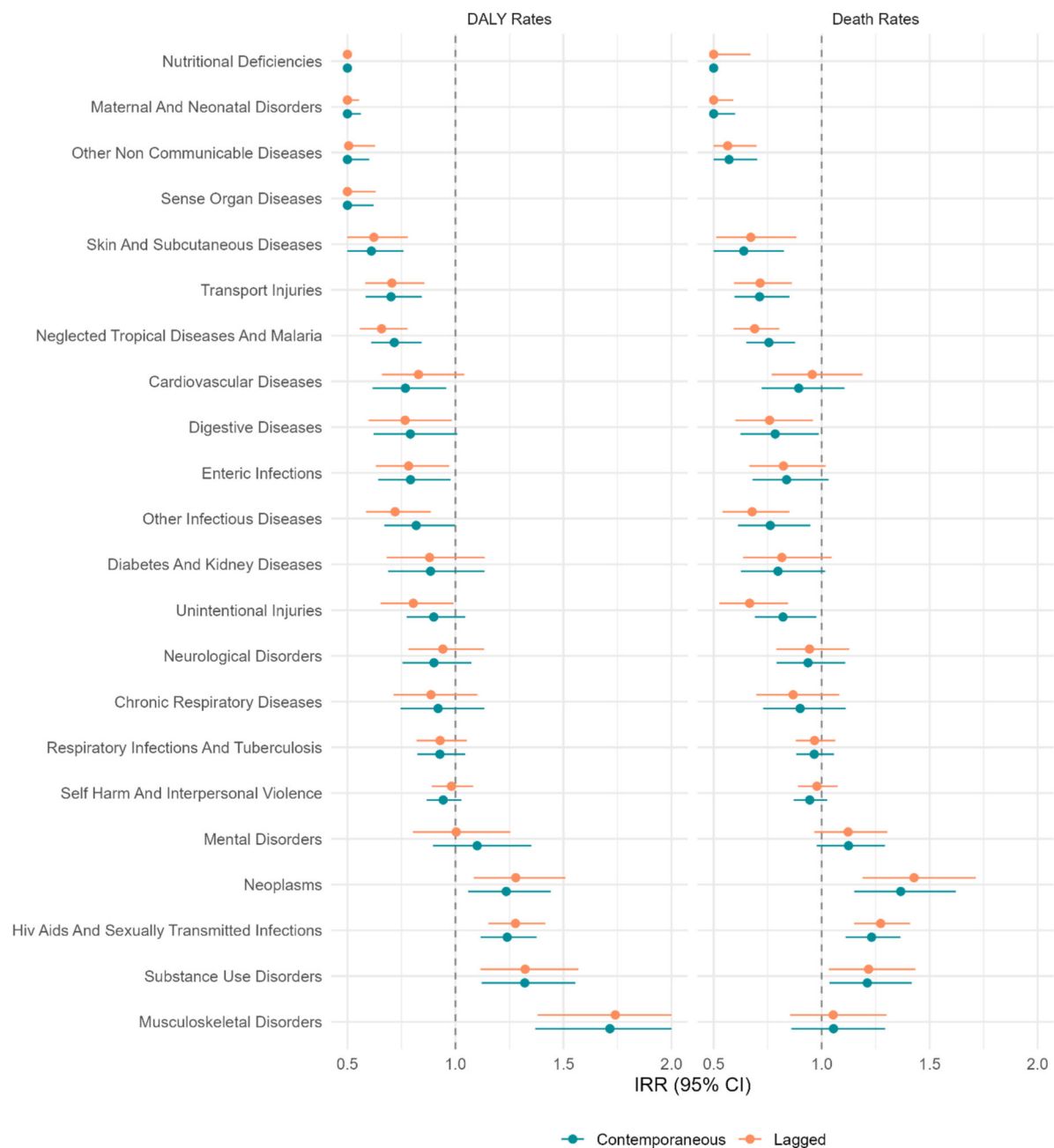


Figure 1. IRRs of health burden association with protest incidence.

maternal and neonatal disorders (0.73, 0.62–0.86, $p < 0.001$), neglected tropical diseases & malaria (0.72, 0.59–0.89, $p = 0.002$), diabetes & kidney diseases (0.70, 0.57–0.86, $p < 0.001$) and nutritional deficiencies (0.47, 0.30–0.74, $p = 0.001$). Several other DALY domains pointed in the same (attenuating) direction but did not reach conventional significance, while neurological burdens showed a weak, non-significant strengthening (1.15, 0.99–1.35, $p = 0.071$). For mortality burdens, attenuation was again common, with significant shifts for substance use disorders (0.84, 0.73–0.98, $p = 0.025$), chronic respiratory diseases (0.83, 0.71–0.97, $p = 0.016$), transport injuries (0.80, 0.68–0.93, $p = 0.005$), other infectious diseases (0.79, 0.63–0.99, $p = 0.044$), other non-communicable diseases (0.76, 0.64–0.91, $p = 0.003$), maternal and neonatal disorders (0.73, 0.62–0.87, $p < 0.001$), digestive diseases (0.73, 0.62–0.86, $p < 0.001$), neglected tropical diseases & malaria (0.68, 0.55–0.84, $p < 0.001$), diabetes & kidney diseases (0.68, 0.55–0.83, $p < 0.001$) and nutritional deficiencies (0.30, 0.13–0.70, $p = 0.006$). No domain exhibited a statistically significant strengthening for either DALY or mortality burdens. These findings are summarised in Figure 3 and Table S13 (Supplementary material).



Figure 2. Random effects (NB2) and fixed effects (Poisson) comparison.

FE Poisson interaction models were estimated as a benchmark (Table S14, Figures 4 and 5). For a subset of causes, the attenuating role of healthcare access was corroborated under FE. Digestive diseases showed convergent attenuation (DALY: RE 0.75, FE 0.49; mortality: RE 0.73, FE 0.55), meaning that both across countries and within countries over time, greater healthcare access was associated with a weaker health–protest link for this cause. Other non-communicable disease mortality also converged (RE 0.76, FE 0.51), as did unintentional injuries (DALY: FE 0.77; mortality: FE 0.75—both significant under FE despite being non-significant under RE). For most other causes, FE interaction estimates pointed in the same attenuating direction as RE but were wider and non-significant, consistent with limited within-country co-variation between health burden changes and access changes over time. Notably, for NTDs and malaria, the RE attenuation (DALY: 0.72; mortality: 0.68) was not replicated under FE (DALY: 1.03; mortality: 1.14), indicating that this moderating effect was primarily cross-national. A similar pattern was observed for transport injuries (RE: 0.80; FE: 0.95).

A pooled three-way model (health $\times$ access $\times$ income tertile) further explored whether the moderating role of healthcare access varied by national income. Greater healthcare access generally weakened the health–protest association most clearly in middle- and high-income settings and for burdens plausibly most amenable to basic services and primary care. These results are reported in Supplementary material (Tables S15, Figures S2 and 3).

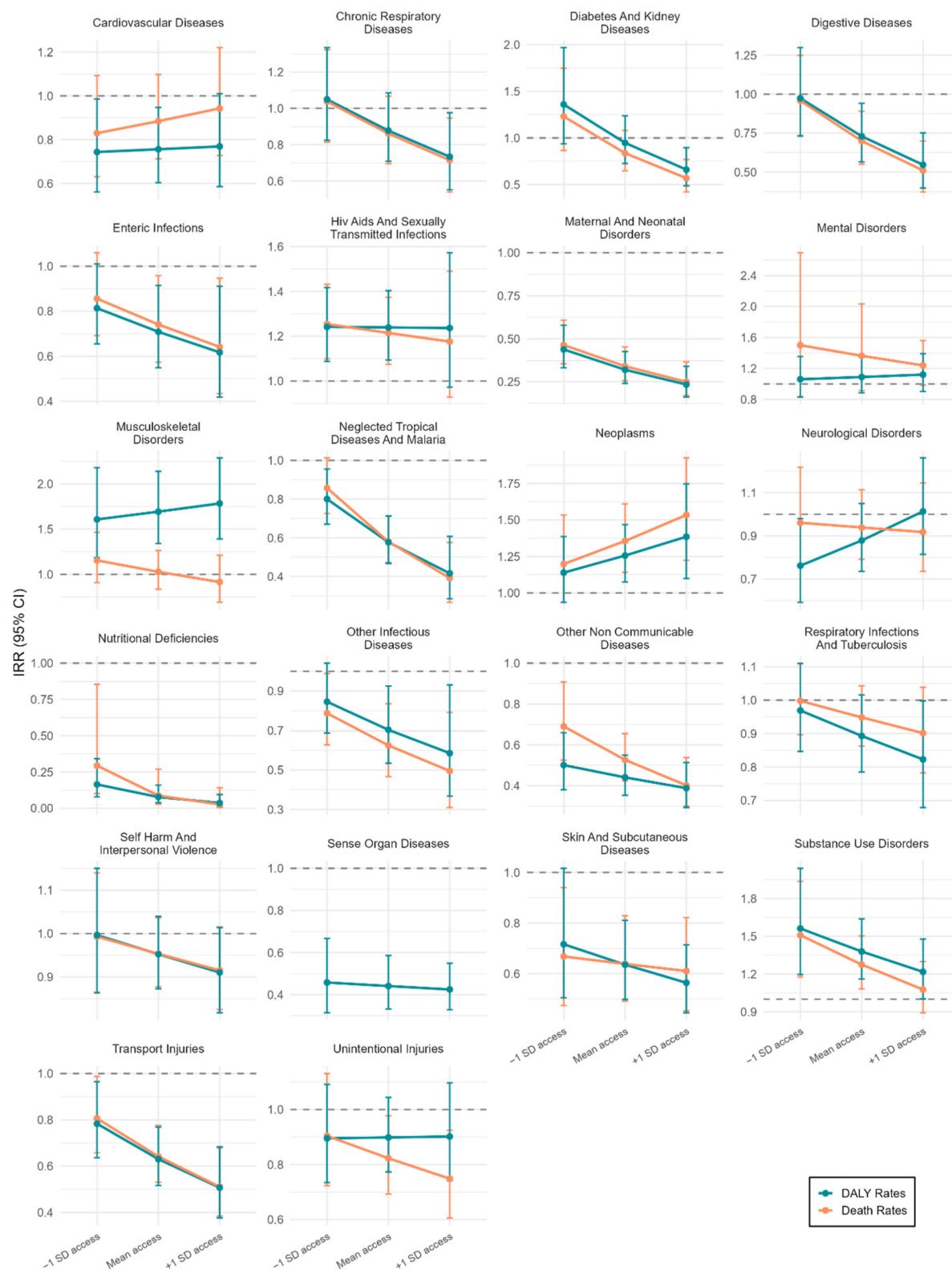


Figure 3. IRR of protest events for each burden by level of healthcare access.

Model diagnostics

Diagnostics did not reveal substantive violations of model assumptions. Across the main-effect models, the pooled health $\times$ access models and the pooled three-way health $\times$ access $\times$ income models ($n = 46$), DHARMA over-dispersion and zero-inflation tests were non-significant in every case (all $p > 0.05$), and all models

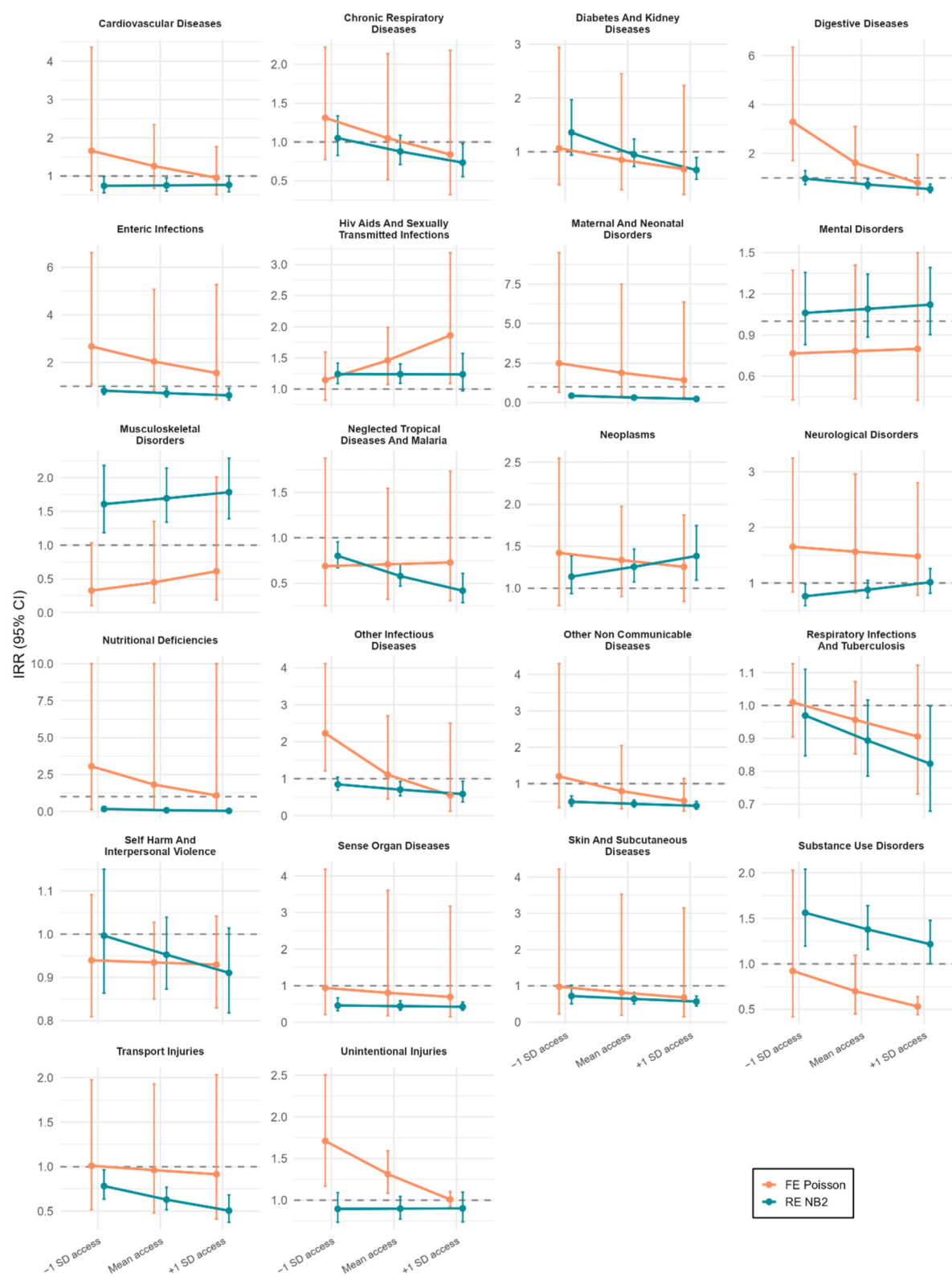


Figure 4. Healthcare access. Random effects (NB2) and fixed effects (Poisson) comparison (DALY).

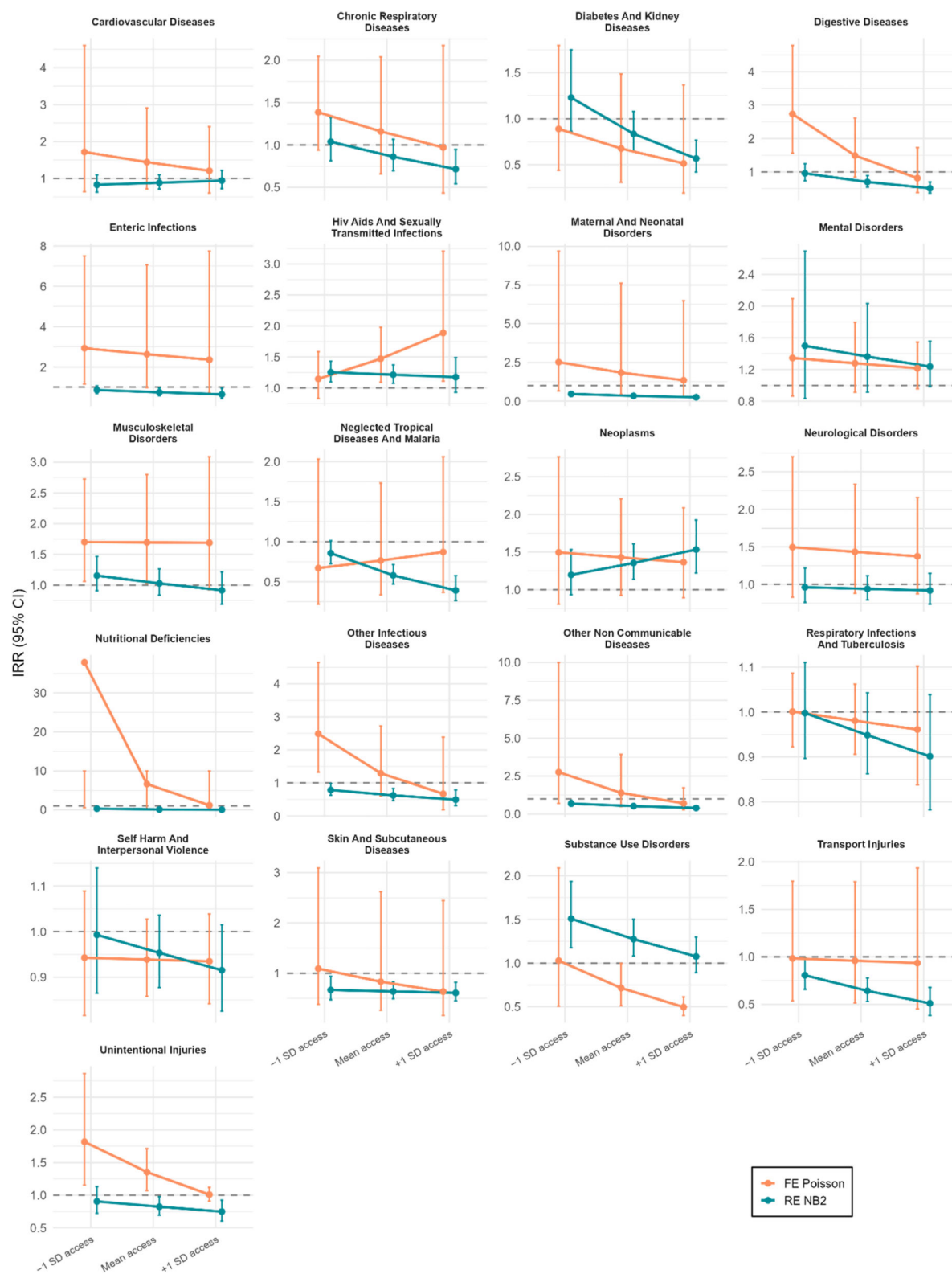


Figure 5. Healthcare access. Random effects (NB2) and fixed effects (Poisson) comparison (mortality).

converged. As is common with large, unbalanced country-year panels, the DHARMA Kolmogorov–Smirnov uniformity test was almost universally significant ($p < 0.001$). Full diagnostics for every model are reported in Table S16 (Supplementary material).

Sensitivity and robustness

The battery of sensitivity and robustness checks described in the methods is reported in full in Supplementary material (Tables S17–22). We summarise the key findings here. Across the four alternative specifications (random-intercept NB, drop-top-1% FE Poisson, zero-inflated NB and post-2010 FE Poisson), robustness varied by cause. Only a minority of causes showed consistent directionality across all four. Neoplasms (both DALY and death rates) were the most robust, with all four specifications yielding IRR > 1 . Other NCDs and NTDs/malaria were consistently negative across most specifications. For many causes, however, the RE-based specifications (random-intercept NB, ZINB) and the FE-based specifications (drop-top-1% FE, post-2010 FE) pointed in opposite directions, consistent with the main RE vs FE comparison and confirming that the source of variation (between- vs within-country) is consequential for inference (Table S18). Results from a restricted sample of countries with stable ACLED coverage ($\geq 80\%$ of analysis years) were broadly consistent with the full-panel RE estimates (Table S21). A reduced specification excluding the Gini index produced nearly identical results (Table S20). Sensitivity models that systematically varied covariate breadth (bivariate through full) showed that the primary associations were stable to the introduction of additional controls (Table S17).

Discussion

This study sought to examine the extent to which population-level health burdens were associated with the incidence of protest events and whether access to healthcare and country income modified these associations. The analyses reveal a divergent pattern. Burdens that cluster in contexts of poverty and early-life risk (for example, maternal and neonatal disorders, nutritional deficiencies, neglected tropical diseases and a range of infectious causes) were associated with lower protest incidence in cross-national (RE) models. By contrast, several chronic and non-communicable burdens, especially musculoskeletal disorders, neoplasms, substance use and HIV/STIs, were associated with higher protest incidence. The FE benchmark revealed that several associations, particularly the negative associations for poverty-linked burdens, were driven primarily by cross-national structural differences rather than within-country temporal dynamics. The strongest evidence emerged for causes where RE and FE estimates converged, namely, HIV/AIDS and neoplasms showed consistent positive associations both across countries and within countries over time, while the moderating role of healthcare access was most robustly confirmed for digestive diseases and unintentional injuries. Importantly, these associations were conditional, with greater access to healthcare generally associated with attenuation of protest mobilisation. Country income further modulated these dynamics. Greater healthcare access generally weakened the association, most clearly in middle and high-income settings.

Several patterns in these findings warrant careful interpretation at the appropriate level of inference. At the country level, this study identifies two broad clusters of association: chronic and non-communicable burdens (musculoskeletal, neoplasms, substance use, HIV/AIDS) were associated with higher protest incidence, while poverty-linked and early-life burdens (maternal/neonatal, nutritional deficiencies, NTDs/malaria) were associated with lower protest incidence. However, the FE analysis revealed that not all of these associations reflected within-country dynamics. For HIV/AIDS and neoplasms, the association held both across and within countries, providing the strongest country-level evidence. For musculoskeletal disorders (RE 1.72, FE 0.46) and substance use (RE 1.32, FE 0.70), the associations were primarily cross-national and may reflect the fact that these burdens were more prevalent in higher-development settings where protest infrastructure, media coverage and political opportunity structures also support higher recorded protest incidence.

Individual-level studies from other settings offer plausible mechanisms for the associations observed here, though ecological data cannot test these directly. Research suggests that chronic health conditions may generate grievances and erode trust in institutions, motivating non-institutional political action such as protest (Kavanagh & Menon, 2024; Mattila, 2020). The association between HIV/AIDS burden and protest is consistent with the well-documented political mobilisation of HIV-affected communities (Berlanda et al., 2024).

Conversely, individual-level evidence suggests that severe deprivation-linked ill-health may deplete the resources and capacity needed for collective action (Anderson et al., 2022). However, we emphasise that these individual-level mechanisms are drawn from other literatures and are not directly tested in this analysis. Alternative macro-level explanations should also be considered. The negative associations for poverty-linked burdens may partly reflect weaker protest event capture in settings with restricted media, limited monitoring infrastructure and higher state repression, factors that correlate with both high deprivation burdens and low recorded protest counts. Similarly, some positive associations may reflect broader development and urbanisation gradients. Notably, countries with higher musculoskeletal or neoplasm burden may be wealthier, and better covered by event-monitoring systems, all of which independently increase recorded protest. The analyses undertaken above helps distinguish these possibilities but cannot fully resolve them, particularly for slow-moving burden measures with limited within-country temporal variation.

The role of healthcare in moderating the health-protest relationship also warrants discussion. Our results suggest that generally, and with some exceptions, greater healthcare access was associated with weaker health-protest associations. That is, greater access to healthcare was associated with fewer protests in relation to a range of health burdens. The FE interaction models confirmed this moderating role for digestive diseases and unintentional injuries, where attenuation was observed both across countries and within countries over time. While for many burdens attenuation was directionally consistent, for many other burdens this suggests that the moderation was primarily cross-national, suggesting it partly reflects development gradients rather than a direct buffering effect of healthcare access. These findings are consistent with the literature that suggests that health and healthcare institutions have a role in bolstering public trust and political engagement. That is, trust in public institutions like healthcare systems are often closely associated with trust in government (Kavanagh & Menon, 2024). For example, amongst a study examining 38 low and middle income countries, poor evaluations of health system performance were associated with lower trust in government (Rockers et al., 2012). Similar findings have been reported in a study that examined 19 European countries, with lower levels of trust in healthcare systems associated with lower trust in political systems (Mattila & Rapeli, 2018). For many, healthcare systems are seen as a primary responsibility of government, and for many, it is the place where they interact with government the most (Menon et al., 2025), it is understandable then, that well-resourced and equitable systems are likely to dampen grievances, as opposed to those that are poorly resourced and inaccessible. It also explains why, where institutions are strong and accessible, including healthcare systems, people are more likely to engage in institutional forms of political action and not protest for example (Mattila, 2020). Importantly, these findings extend beyond Europe and beyond the provision of healthcare itself. For example, the successful expansion of anti-retroviral therapy (ART) was associated with reduced social conflict in a number of African countries (Berlanda et al., 2024), the mechanism for which could be theorised in a similar way.

Notably, when it comes to healthcare access our findings also align closely with, and extend, the literature on amenable mortality, that is, the causes for which deaths (and, by implication, a substantial share of disability) should not occur given timely and effective health care. Several previous studies have identified communicable, maternal, neonatal and nutritional diseases as conditions for which mortality is amenable to healthcare (Haakenstad et al., 2022; Nolte & McKee, 2004). In our models, in many cases, it was these amenable burdens where expanded access to healthcare was associated with the greatest suppressions in protest mobilisation.

These results speak directly to, and help to nuance, the 'health-gap/politicisation' hypothesis (Anderson et al., 2022). Our results suggest that where health burdens are tightly coupled to absolute deprivation, demobilisation dominates in cross-national comparisons. Put differently, where ill-health is intertwined with deprivation, the health-capacity gap may be too large for politicisation to take hold. However, caution is warranted against interpreting these cross-national patterns as reflecting direct effects of health burdens on protest behaviour. The associations observed here are ecological and may reflect broader development, governance and data-quality gradients that coincide with both health burden profiles and protest capacity. Conversely, many of the burdens that were positively associated with protest were those that generated chronic functional limitations, long-term care needs and sustained contact with health institutions. For HIV/AIDS and neoplasms, the within-country evidence strengthens the case that these burdens are genuinely associated with heightened mobilisation, rather than simply marking higher-development contexts. At the same time, across most burdens, greater access to healthcare was associated with attenuation of health-protest relationship, with greater access

associated with fewer protests. The impact that healthcare access had however also appeared to be shaped by country income and the extent to which the burden in question was amenable to treatment. Together, these results suggest that speaking about ‘health’ when it comes to political engagement may be far too general and at the same time, important factors, like access to healthcare should also be considered in examining the links between health, protest and other forms of political engagement.

Limitations

While the above conclusions are strengthened by extensive diagnostics and robustness checks, several limitations nevertheless warrant caution. First, this was an observational, ecological study of cross-national associations. The relationships detected at the country level may not hold for individuals, movements or communities, a limitation inherent to all ecological analyses. The use of a one-year lag provides a useful temporal ordering but did not establish causality in a strong sense, as many of these health burdens are slow-moving markers of underlying structural context. The divergence between RE and FE estimates for several causes underscores that observed associations may partly reflect broader development and urbanisation patterns or differences in protest event capture, rather than disease-specific political consequences. Second, complete-case analysis means the analytic sample was not representative of all countries. Included country-years were substantially wealthier, more democratic, and had higher protest counts than excluded observations (Table S19). This selection may have attenuated or inflated associations in ways that cannot be fully assessed. We addressed this through a reduced specification, a restricted-sample sensitivity and transparent reporting of included versus excluded characteristics, but residual selection bias remains possible. Third, ACLED’s coverage expanded substantially over the study period (48 country-years in 2004 to 243 by 2021), and event detection likely varied with media openness, state repression, infrastructure and monitoring intensity, factors that may also correlate with health-system performance and political covariates. While we controlled for this in assembling our dataset and tested sensitivity to a stable-coverage subset, care is warranted. The assumption that covered country-years with zero recorded protests reflect true absence of protest activity may not hold in contexts with restricted media or high repression. Fourth, the V-DEM healthcare-equity index, although conceptually robust, is derived from expert assessments not policy itself, which may have influenced these findings. This measure captures expert-assessed health equality across social groups rather than healthcare access or service provision in a narrow sense. Interpretations of the moderation findings should therefore be understood as reflecting expert-assessed health equality rather than direct measures of healthcare access. Fifth, unmeasured influences, such as state repression, social-media reach, internet penetration, unemployment, or transnational advocacy could shape both health conditions and protest activity, potentially confounding observed associations. Future work should aim to link individual health surveys or data with protest event records and to exploit quasi-experimental reforms in health systems to strengthen causal inference. Sixth, the large number of cause-specific models creates a multiple comparisons concern. While we applied corrections and focused interpretation on broad patterns, some individual findings, particularly those that did not survive correction, may reflect chance variation rather than true associations. Seventh, some burden categories are broad and heterogeneous. For example, ‘other non-communicable diseases’ and ‘sense organ diseases’ do not lend themselves to clear interpretation. We focused the interpretive discussion on the clearest categories and caution against over-reading broad residual categories.

Conclusions

This study shows that the political salience of ill-health is contingent rather than uniform: burdens linked to deprivation and early-life risk were associated with lower protest incidence in cross-national comparisons, while chronic, disability-producing conditions were more often associated with mobilisation, and both patterns were shaped by the reach of health systems and national income. The fixed-effects analysis revealed that several associations, particularly the negative associations for poverty-linked burdens, were driven by cross-national structural differences, cautioning against interpreting these as direct effects of health burdens on protest. The strongest evidence emerged for causes where associations held both across and within countries, notably HIV/AIDS and neoplasms. When it came to healthcare access, across almost every burden, the expansion of access

was generally associated with attenuation of the health-protest association, with digestive diseases and unintentional injuries showing convergent evidence under both RE and FE specifications. This is consistent with the view that building equitable, accessible health systems may be associated with reduced grievances, particularly where preventable burdens are concentrated. These findings should be understood as identifying cross-national associations that are consistent with, but do not establish, causal mechanisms linking population health to collective political action. Perhaps the most clear message from these findings is that health is not only political in relation to its determinants; it also has political implications in shaping how and when collective dissent is voiced.

Endnotes

1. We use the term 'burden' throughout to refer to the healthy life years lost due to death or disability, using it interchangeably to refer to both DALYs and mortality in the analysis below.
2. For the purposes of this study we have conceptualised protest using the definition provided by ACLED as 'non-violent demonstrations, involving typically unorganised action by members of society' (ACLED n.d.)
3. In this paper we conceptualise politics broadly, as the exercise of power, that could include a range of actions or activities, protest being one of these.
4. This variable, like most in the V-DEM dataset, was derived from expert-coded assessments on an ordinal scale (e.g. from 'extreme inequality' to 'equal access'), which are then aggregated and converted into a continuous latent index using a Bayesian measurement model, combining multiple country-expert ratings into a comparable country-year score.

Author contributions

CRediT: **Ryan Essex:** Conceptualization, Formal analysis, Methodology, Visualization, Writing – original draft, Writing – review & editing; **Trevor Thompson:** Formal analysis, Writing – original draft, Writing – review & editing; **Sorcha A. Brophy:** Writing – original draft, Writing – review & editing; **Veena Sriram:** Writing – original draft, Writing – review & editing; **Jagnoor Jagnoor:** Writing – original draft, Writing – review & editing; **Rita Issa:** Writing – original draft, Writing – review & editing.

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Data availability statement

All data used in this analysis is publicly available.

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